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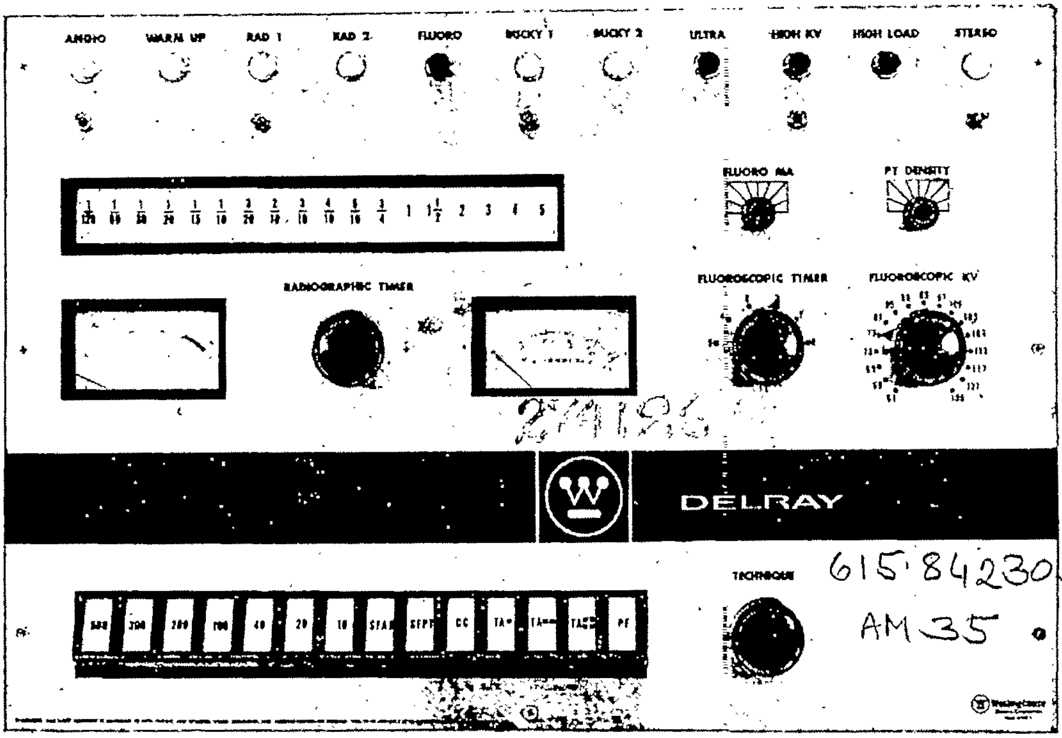

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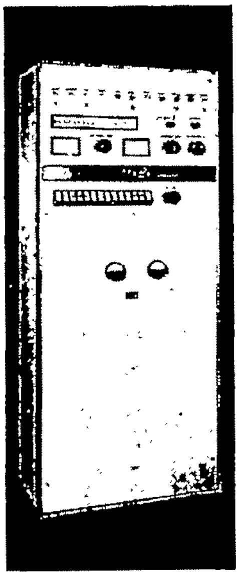

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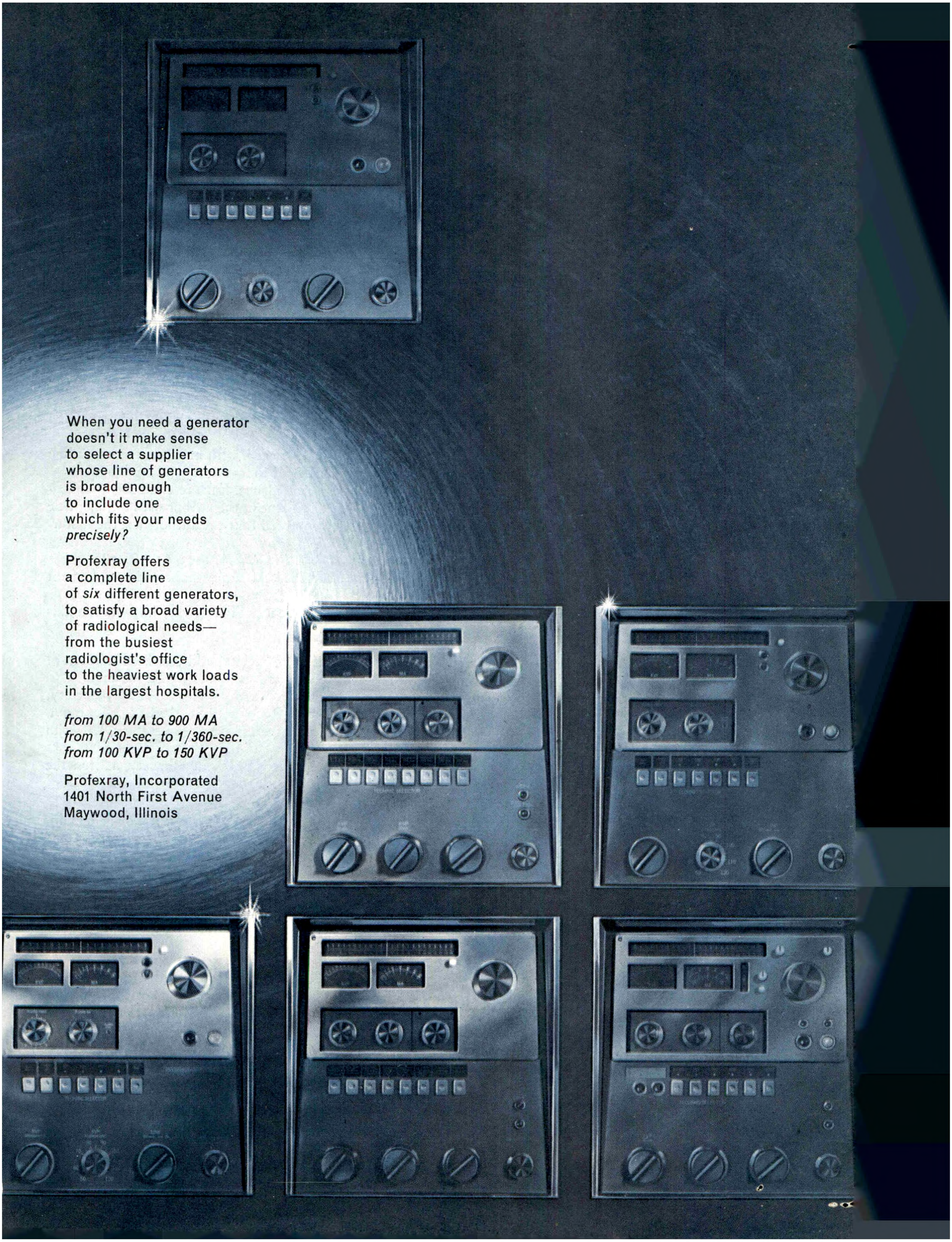

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VOL. 92 OCTOBER, 1964 NO. 4

CONTENTS

Veins of the White Matter of the Cerebral Hemispheres (the Medullary Veins); Diagnostic Importance in Carotid Angiography. YUN PENG HUANG, M.D., AND BERNARD S. WOLF, M.D. 739

Aneurysm of the Vein of Galen; Case Report and Review of the Literature. WARREN RUSSELL, M.D., AND THOMAS H. NEWTON, M.D. 756

The Primitive Trigeminal Artery As Seen Angiographically and at Postmortem Examination. GERTRAUD WOLLSCHLAEGER, M.D., AND PAUL B. WOLLSCHLAEGER, M.D. 761

Giant Cell (Cranial) Arteritis; A Case with Angiographic Abnormalities. VINCENT C. HINCK, M.D., C. CONRAD CARTER, M.D., AND JOHN G. RIPPEY, M.D.. 769

Changes in the Anterior Choroidal Artery in Intracranial Lesions. MARJORIE J. LEMAY, M.D., AND DAVID M. JACKSON, M.B., B.S.. 776

Unilateral Intracranial Hematoma without Shift of the Anterior Cerebral Artery. SANFORD J. LARSON, M.D., LEON LOVE, M.D., AND ALLEN MITTELPUNKT, M.D. 786

Angiographic Features of Unilateral Hydrocephalus of Obstructive Nature. YUN PENG HUANG, M.D., AND BERNARD S. WOLF, M.D. 792

Evaluation of Angiography in the Diagnosis of Suprasellar Tumors. EMANUEL H. FEIRING, M.D., AND JEROME H. SHAPIRO, M.D. 811

Percutaneous Retrograde Brachial Angiography in the Diagnosis of Acoustic Neurinoma; Results in 4 Cases. JOHN A. GOREE, M.D., GEORGE T. TINDALL, M.D., AND GUY L. ODOM, M.D. 829

A Case of Aneurysmal Malformation of the Vertebral and Basilar Arteries Causing Cranial Nerve Involvement. MICHAEL SCOTT, M.D., AND HERBERT M. STAUFFER, M.D. 836

Subdural Empyema; Report of a Case Demonstrating the Unusual Angiographic Triad. ERNEST J. FERRIS, M.D., AND JULIUS CIEMBRONIEWICZ, M.D. 838

(*Continued on page iv*)

The Preoperative Diagnosis of Brain Abscess by the Roentgenologic Demonstration of an Air-Fluid Level; A Report of Two Cases. Judah Zizmor, M.D., Arnold M. Noyek, M.D., Richard J. Bellucci, M.D., Morton L. Gutkin, M.D., and Edmund Vermes, M.D. 844

Angiographic Demonstration of Bilateral Carotid Body Tumors. Morton A. Bosniak, M.D., Bernard Seidenberg, M.D., Ira C. Rubin, M.D., and Sidney Arbeit, M.D. 850

Aberrant Right Subclavian Artery; Dispute of Commonly Accepted Concepts. John W. Beabout, M.D., James R. Stewart, M.D., and Owings W. Kincaid, M.D. 855

Calcification in the Coronary Arteries and Its Relationship to Arteriosclerosis and Myocardial Infarction. William G. Beadenkopf, M.D., Assaad S. Daoud, M.D., and Blanche M. Love, B.Sc. 865

Coronary Arteriography; Some Observations on Technique and Interpretation. Manuel Viamonte, Jr., M.D., Arthur J. Gosselin, M.D., and Leonard S. Sommer, M.D. 872

Thrombotic Occlusion of the Abominal Aorta; Seven Year Angiographic Study of a Case. Robert M. Schmidt, M.D., Jack W. C. Hagstrom, M.D., and Israel Steinberg, M.D. 877

Aortic Atresia without Significant Hypoplasia of the Ascending Aorta. Waldo López, M.D. 888

Intravenous Angiocardiography, Abdominal Aortography, and Peripheral Arteriography with Single Arm Pressure Injection. Israel Steinberg, M.D., and Harry L. Stein, M.D. 893

Angiographic Artefacts Which Simulate or Mask Abnormality. Walter W. Herbert, M.D. 907

Percutaneous Transfemoral Arteriography; An Analysis of the Complications in 1,000 Consecutive Cases. Mordecai Halpern, M.D. 918

An Arterial Simulator for Angiographic Research. Fred C. Shipps, M.D. . 935

Programmed Angiography. Fred C. Shipps, M.D. 939

Successful Sterilization of a Primary Cardiac Sarcoma by Supervoltage Radiation Therapy. Robert H. Sagerman, M.D., Edward Hurley, M.D., and Malcolm A. Bagshaw, M.D. 942

Officers 947

Editorial

The Evolution of Cerebral Arteriography. John Gilroy, M.D. . . . 948

National Council on Radiation Protection and Measurements (NCRP) . . 951

The Eleventh International Congress of Radiology 953

News Items 955

Books Received 956

Abstracts of Radiological Literature 957

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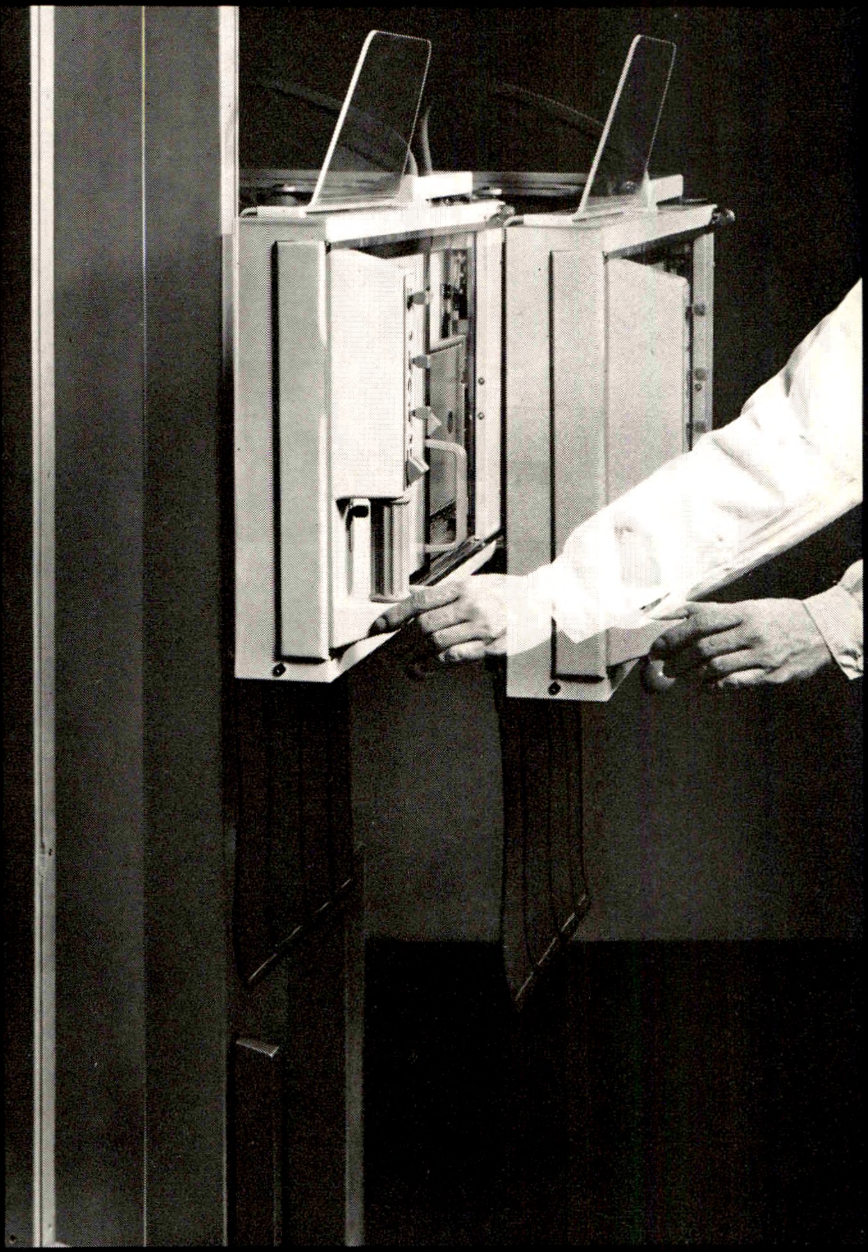

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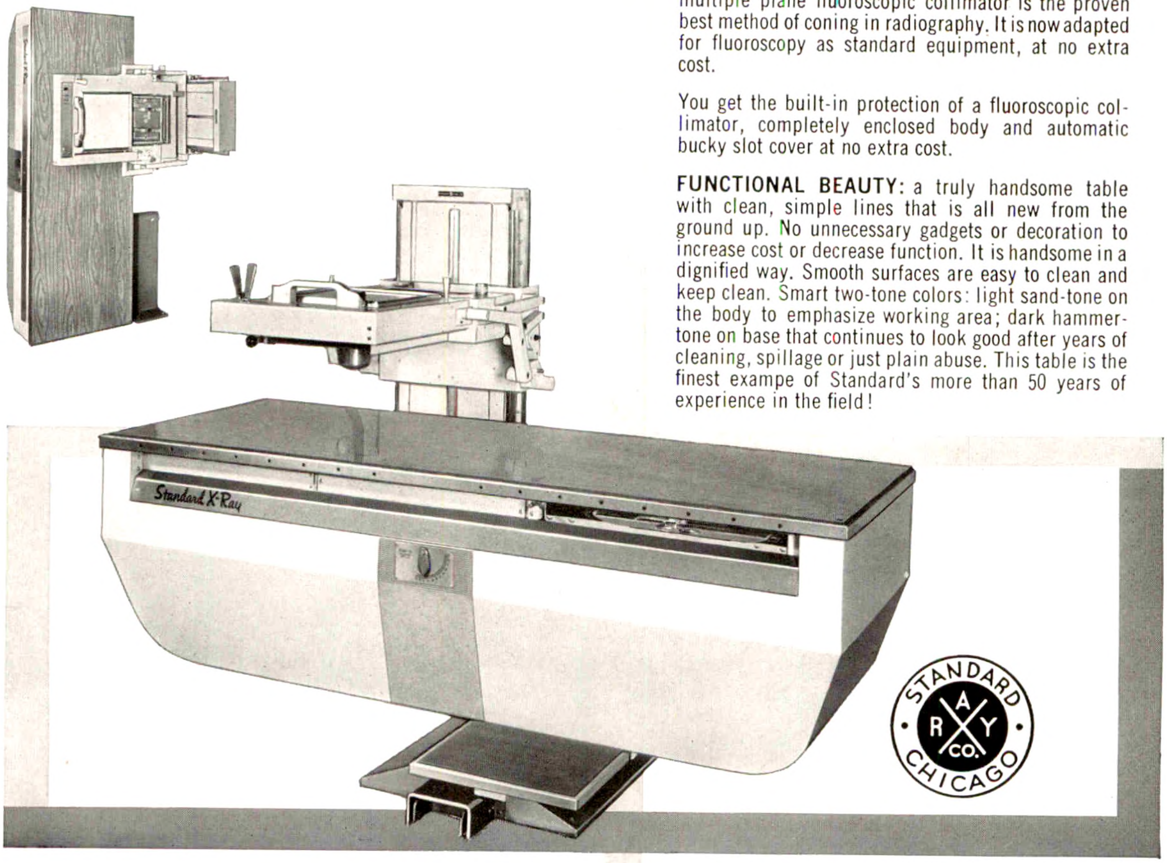

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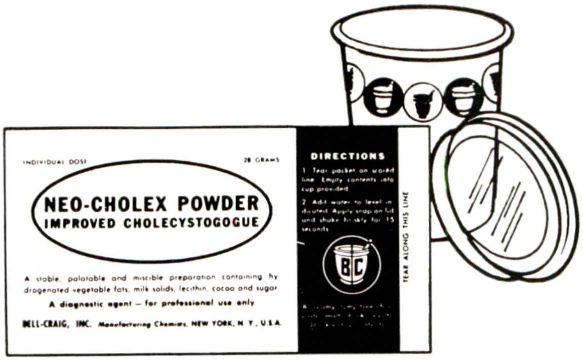
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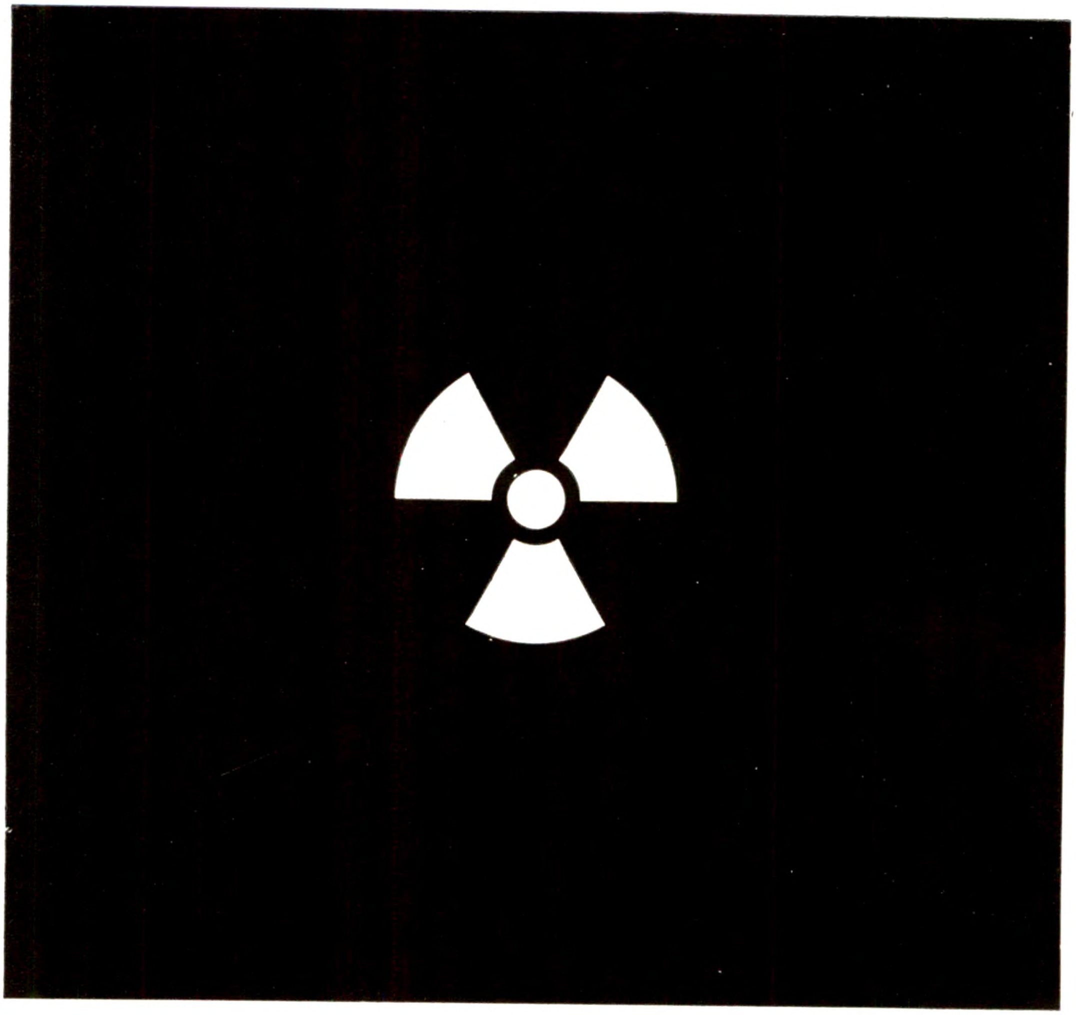

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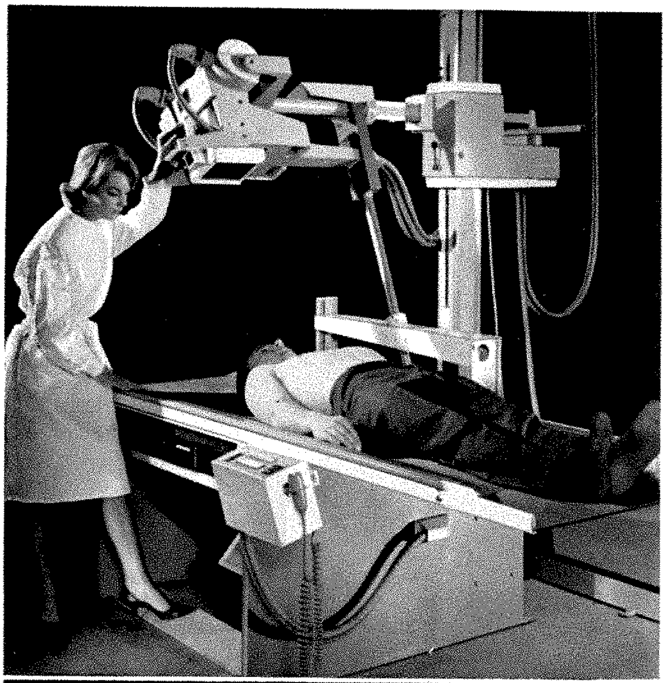

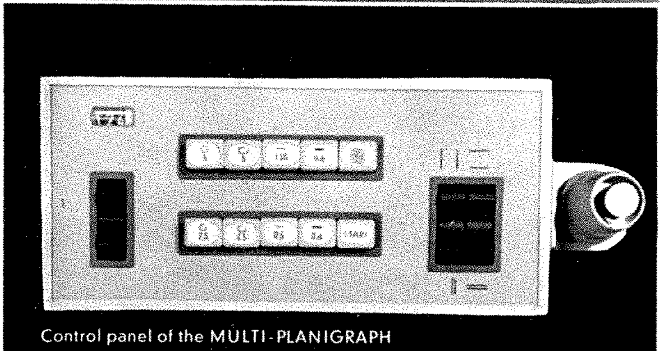

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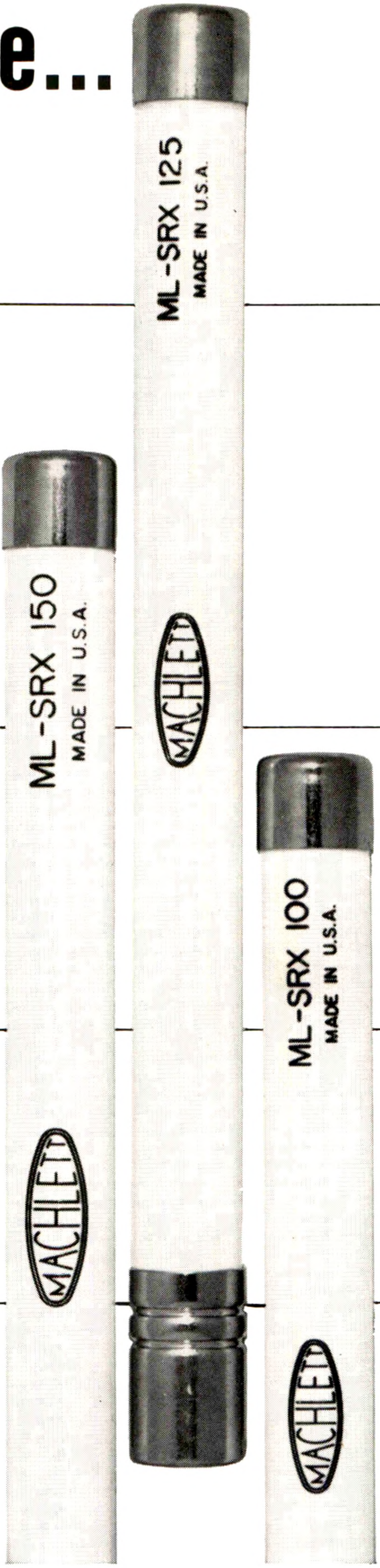

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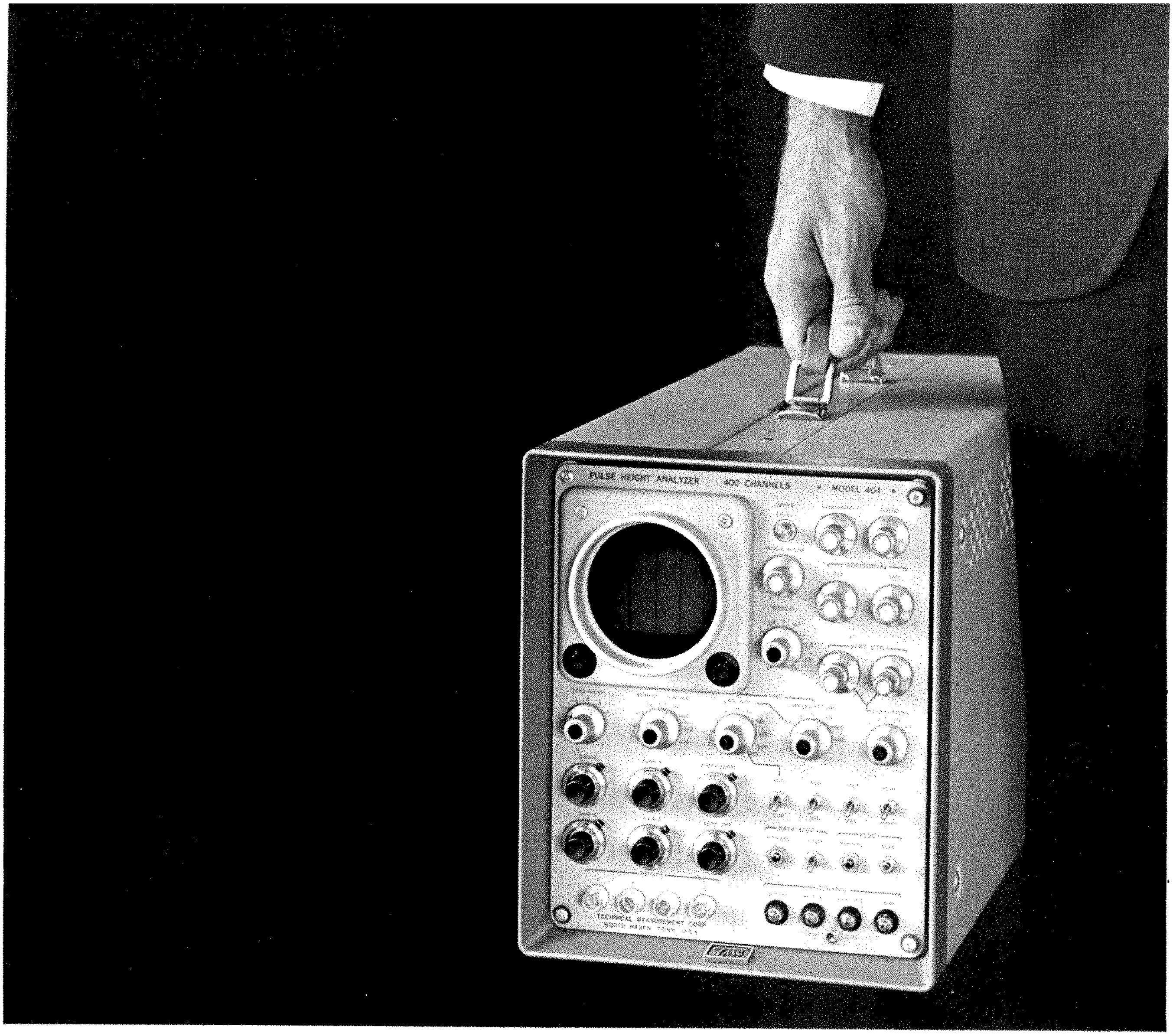

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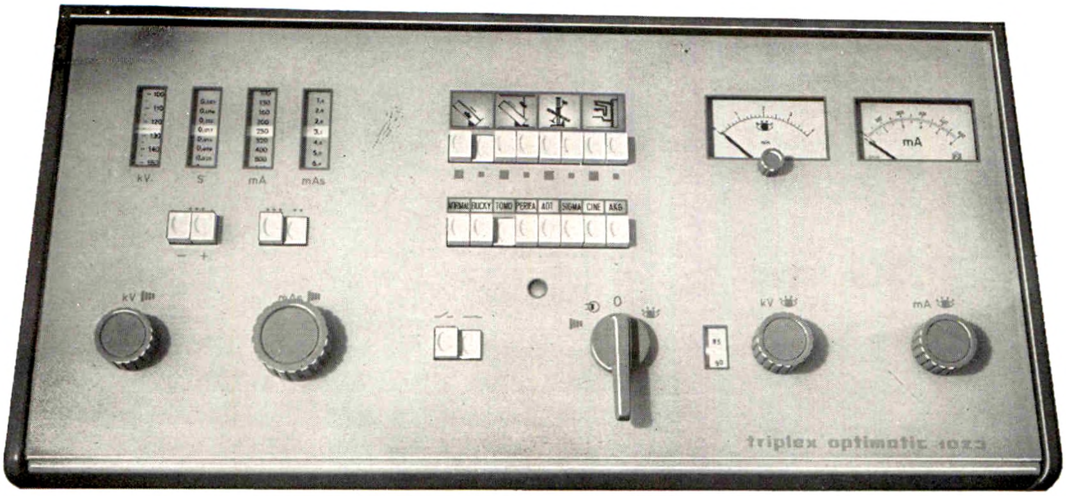

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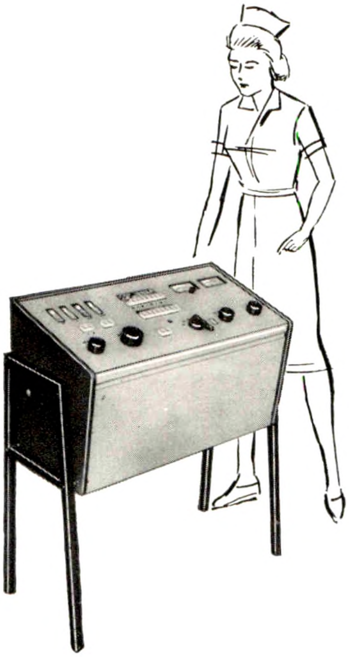

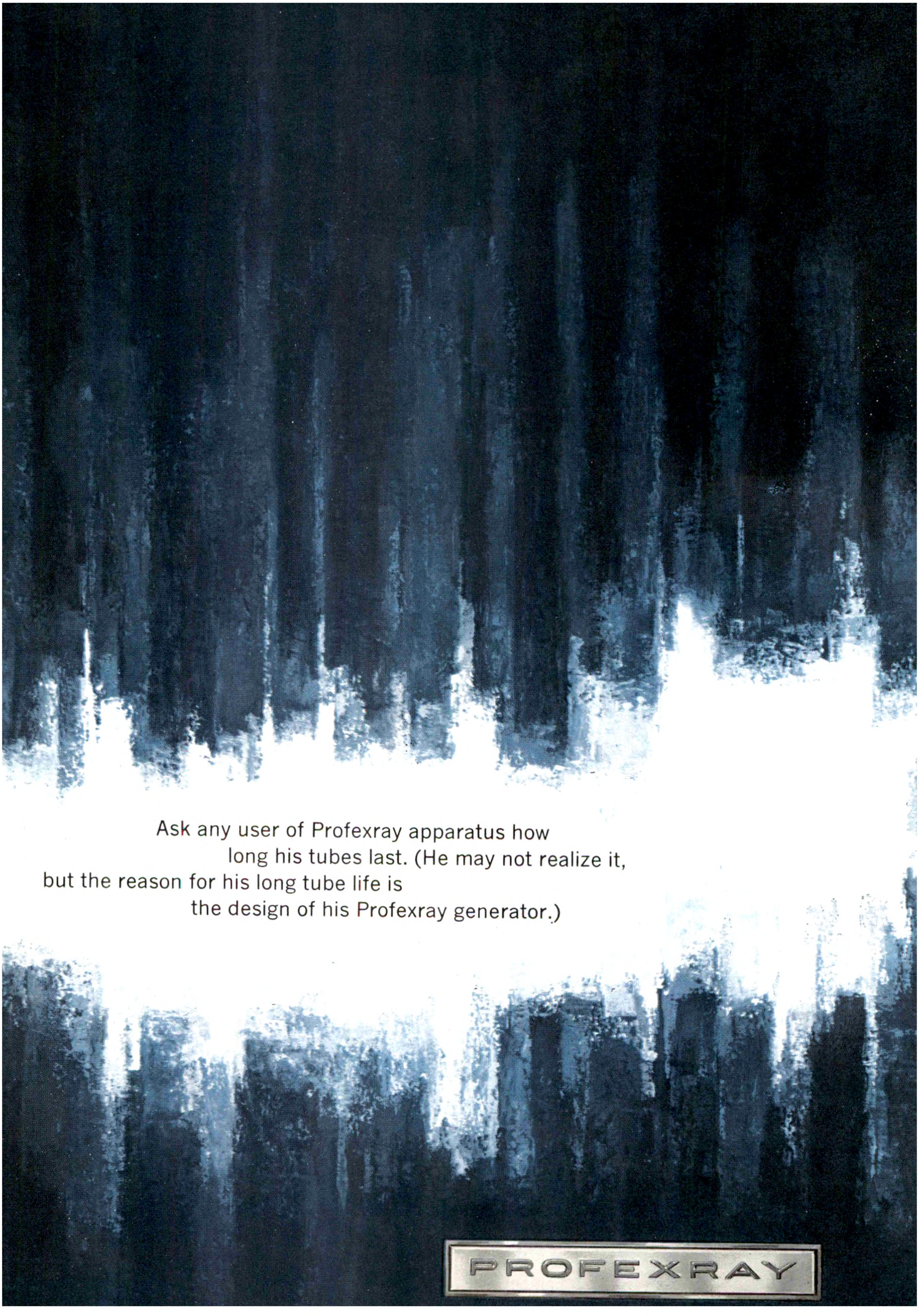

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VOL. 92 OCTOBER, 1964 No. 4

VEINS OF THE WHITE MATTER OF THE CEREBRAL HEMISPHERES (THE MEDULLARY VEINS)*

DIAGNOSTIC IMPORTANCE IN CAROTID ANGIOGRAPHY

By YUN PENG HUANG, M.D., *and* BERNARD S. WOLF, M.D.
NEW YORK, NEW YORK

THE angiographic appearances of the subependymal tributaries of the internal cerebral vein and of the basal vein of Rosenthal have been previously described and a classification of these vessels into medial and lateral groups has been suggested.[16] The recognition of these veins is of diagnostic significance as they serve to outline portions of the walls of the lateral ventricles. The veins within the cerebral hemispheres which drain into the subependymal veins are rarely evident because of their small size. There are, however, circumstances under which these vessels become large enough to be easily seen on angiography. It is therefore of importance to understand the nature and distribution of these intracerebral veins.

The veins within the substance of the cerebral hemispheres can be divided into two main categories. One category, the striate veins, serves to drain the basal ganglia and has been described in the anatomic literature in some detail.[4,9,12] The primary interest of the present report is in the second category of intracerebral veins, namely, those within the white matter of the cerebral hemispheres. There has been no name generally adopted to designate these veins. The anatomic description by Duret[1] and by Pfeiffer[8] refers to these vessels as "medullary veins" and this designation appears sufficiently apt to be recommended for general usage. While many features of these veins have not been completely delineated because of the difficulties of anatomic studies, the medullary veins consist essentially of two groups (Fig. 1). One of these includes the short venous channels which begin in the white matter 1 or 2 cm. below the cortical gray matter and run directly through the cortex to join the pial veins on the surface of the cortex. As these veins traverse the cortical gray matter, they participate in the drainage of the gray matter. However, the bulk of the venous drainage of the cortical gray matter is via innumerable microscopic intracortical veins which enter the superficial pial veins directly. The second and more prominent group of medullary veins consists of a large number of relatively long venous channels which originate approximately in the same region of the white matter as the first group but run deeply and converge towards the lateral ventricle. These veins drain via the subependymal veins into the internal cerebral vein or the basal vein of Rosenthal. It is convenient to designate the first group of medullary veins as "superficial medullary veins" and the second as "deep medullary veins." There is a small overlapping or junctional area in the white matter a short distance from the cortex which may be drained either superficially or deeply. In addition to these main groups, there are also said to be venous channels which traverse the entire thickness of the cerebral hemisphere and connect superficial cortical veins with subependymal veins. Veins such as these have been called by Schlesinger[12] "intracerebral anastomotic veins" and by Kaplan[5] "transcerebral veins." Padget[7] described in a 40 mm. human embryo "many notably straight veins which pass directly laterally from the subependymal layer to the external surface of the brain." However, there is no agreement as to the frequency and importance of such vessels in the fully developed brain. It is likely that, more commonly, connections between the superficial and deep medullary veins are by anastomotic veins which bridge these vessels or simply by a venous plexus.

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On gross examination of the brain and on angiography, it is difficult to recognize the superficial medullary veins since they are barely visible to the naked eye. Low power views of microscopic sections demonstrate that these vessels have a surprisingly straight course as they proceed toward and

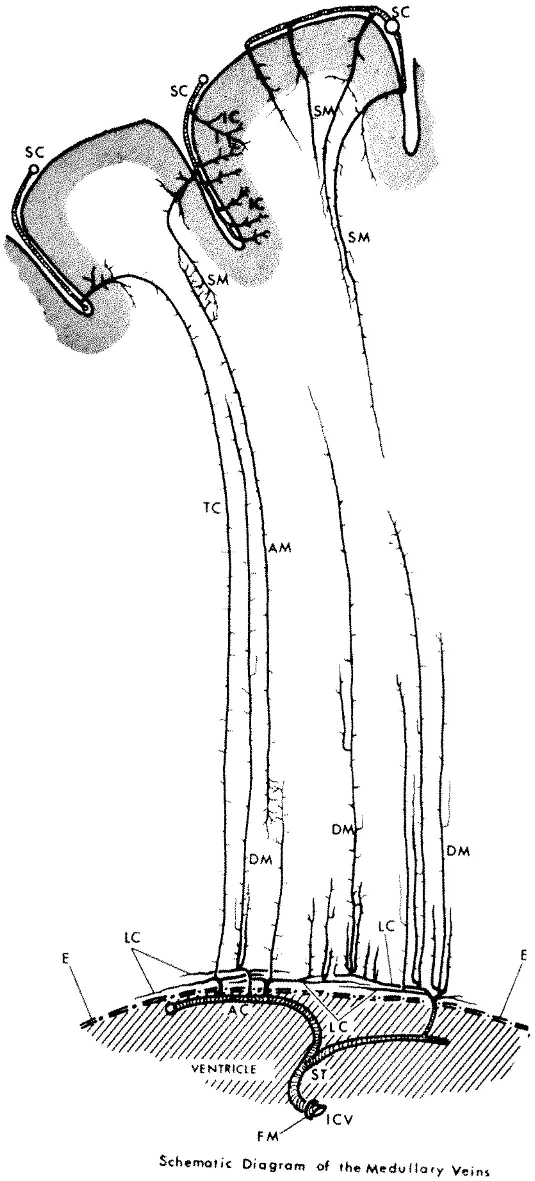

Fig. 1. Diagram of the medullary veins in relation to the superficial cortical and subependymal veins. The venous drainage of the cortical gray matter (shaded) occurs primarily via small intracortical veins (IC). The superficial medullary veins (SM) arise in the juxtacortical white matter and penetrate the gray matter in order to join the superficial cortical veins (SC). The deep medullary veins (DM) are of variable length. Many of these originate near the juxtacortical white matter and therefore are considerably longer than the superficial medullary veins. The deep medullary veins are shown draining into longitudinal caudate veins (LC) along the upper outer angle of the lateral ventricle. These vessels then drain into subependymal trunks, the anterior caudate vein (AC) and the striothalamic vein (ST). A "transcerebral vein" (TC) is also illustrated extending from a superficial cortical vein to a subependymal vein. Also illustrated is a long "anastomotic medullary vein" (AM) intervening between a superficial medullary vein and a short deep medullary vein. Other labeled structures include the internal cerebral vein (ICV), the foramen of Monro (FM), and the ependyma (E).

through the cortex. The veins of this group which perforate the convex portions of the gyri, continue this straight course and enter a superficial cortical vein at a right angle. Others arch laterally before reaching the surface to enter a pial vein in the depth or along the side walls of sulci. While some superficial medullary veins may be large enough to be seen normally on angiography, it is difficult to detect any clear-cut boundary between such vessels and the small surface veins over the gyri and in the sulci.

The deep medullary veins are larger than the superficial medullary veins and many are evident on gross examination of sections of the brain. In order to demonstrate their full extent and number, however, special techniques, *e.g.*, injection with colored or radiopaque materials, are required. Such studies indicate that these vessels often follow the course of the radiations of the corpus callosum, specifically the forceps major, the forceps minor, the corona radiata and the tapetum. However, deviations from this pattern particularly towards their terminations are common. These vessels are of small caliber but disproportionately long with a straight course as they run deeply towards the lateral ventricle. There is also considerable disproportion between these main channels and their tributaries which are numerous but microscopic in size. These microscopic tributaries join the main channels almost at a right angle.[12] This arrangement is in contrast to venous drainage systems in other viscera which form trunks of gradually increasing caliber by progressive union of small tributaries. As the deep medullary veins converge towards the ventricle, they increase relatively little in caliber towards their terminations. As a group, they form a characteristic wedge-shaped or fan pattern (Fig. 2, *A* and *B*). Individual vessels may join one or another of the subependymal veins directly. On the other hand, many of the deep medullary veins which converge towards the body of the lateral ventricle make a right angle turn along the superior lateral corner of the ventricle and run parallel to this corner for some distance before entering a subependymal trunk (Fig. 1 and 2, *A* and *B*). These veins were designated by Schlesinger[12] as "longitudinal caudate veins." Longitudinal vessels similar to these are also common in the anterolateral corner of the frontal horn and may rarely be seen in the roof of the atrium and posterior horn.

The deep medullary veins do not converge to the walls of the lateral ventricle in random fashion but are concentrated in particular areas. By far the most constant of these areas can be visualized as a band or line above the head and body of the caudate nucleus corresponding to the location of the "longitudinal caudate veins" noted above. As this band or line approaches the atrium, it bifurcates and extends posteriorly into the roof of the occipital horn and inferiorly into the temporal horn along the lateral aspect of the tail of the caudate nucleus. This band toward which the deep medullary veins converge corresponds to the regions in which the original "subependymal gray matter" persists in the fully developed brain. The reason for this can be surmised from a consideration of the embryologic development of the cerebral hemisphere.

The original telencephalic vesicles are thin-walled structures with a complete lining of primitive neuroectodermal cells. During embryologic development, cells migrate from their original subependymal location to the surface to form the primary cerebral cortex. As these cells migrate, the subependymal venous plexus continues to serve as the chief venous drainage pathway for the intervening developing white matter. With the intrusion of the thalamus, basal ganglia, corpus callosum and fornix into the walls of the lateral ventricle, the originally continuous subependymal gray matter becomes confined to the narrow band-like area described above. As a result, the terminations of the medullary veins also become concentrated and converge toward these localized areas. The fact that proliferation and migration of cells are most active lateral and dorsal to the body of the caudate nucleus[13] may serve to explain the more numerous deep medullary veins in the posterior frontal and parietal areas.

The appearance of the deep medullary veins in the different lobes of the hemisphere (Fig. 2, *A* and *B*) is determined to a considerable extent by the shape of the lobe and the portion of the ventricular system within it. The medullary veins from the frontal pole run backward toward the anterior horn and drain primarily into tributaries of the vein of the septum pellucidum. The deep medullary veins in the remainder of the frontal lobe and in the anterior parietal region run downward and medially toward the superior and lateral corner of the frontal horn and body of the lateral ventricle. These vessels drain into the tributaries of the striothalamic vein and the direct lateral subependymal vein. The deep medullary veins in the posterior parietal and occipital areas run forward and medially to the roof and lateral wall of the atrium and join medial and lateral atrial subependymal veins. The veins from the posterior portion of the temporal lobe run upward and forward to join the lateral atrial and inferior ventricular subependymal veins. The deep medullary veins from the anterior temporal lobe run backward to join the subependymal tip veins of the temporal horn and, via these veins, the basal

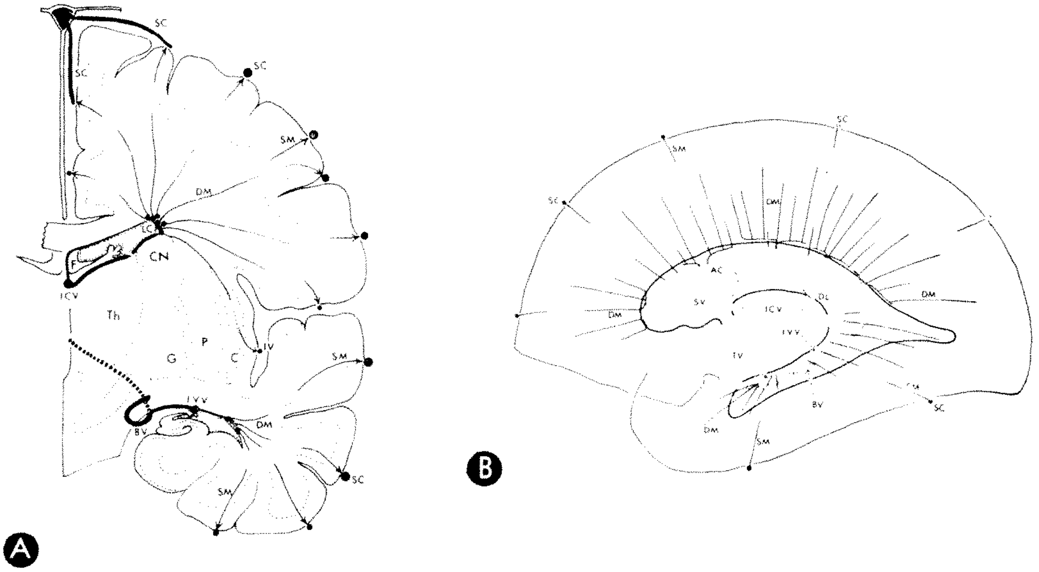

FIG. 2. Schematic diagram of the medullary veins.

(*A*) Coronal section of the brain through the body of the lateral ventricle. The short superficial medullary veins (SM) extend to the surface and join superficial cortical veins (SC). The veins of the insula (IV) are part of the superficial cortical system. The deep medullary veins (DM) drain into subependymal veins of the body of the ventricle and temporal horn. The deep medullary veins in the parasagittal and suprasylvian area show a characteristic converging pattern as they drain towards the outer angle of the body of the lateral ventricle to join the longitudinal caudate veins (LC). The deep medullary veins of the temporal lobe are smaller and converge to subependymal veins in the roof of the temporal horn and, via these vessels, into the inferior ventricular vein (IVV). This latter vein joins the basal vein (BV). Other labeled structures include the fornix (F), the internal cerebral vein (ICV), the thalamus (Th), the caudate nucleus (CN), the globus pallidus (G), the putamen (P), and the claustrum (C).

(*B*) Lateral view. The deep medullary veins (DM) run toward the superior lateral corner of the lateral ventricle and drain into the septal vein (SV), the anterior caudate vein (AC), the direct lateral vein (DL), the tip veins of the temporal horn (TV) and the inferior ventricular vein (IVV). The line of junction between the deep medullary veins and the subependymal venous trunks serves as a landmark to indicate the border of the lateral ventricle. Other structures shown are the internal cerebral vein (ICV), the basal vein of Rosenthal (BV), superficial medullary (SM) and superficial cortical (SC) veins.

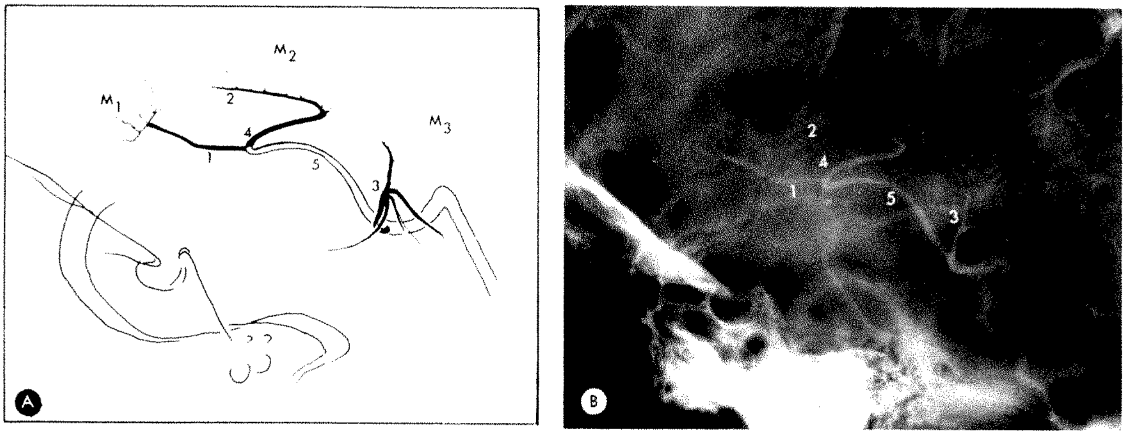

Fig. 3. Normal appearance.

(*A*) Tracing of lateral view. The stems of numerous deep medullary veins are seen as thin faint vessels joining the larger subependymal veins. The anterior group (M_1) enters the vein of the septum pellucidum (1) along the anterior lateral border of the frontal horn. The frontoparietal medullary veins (M_2) enter the anterior caudate vein (2) along the superior lateral border of the body of the ventricle. The parietal medullary veins (M_3) enter the direct lateral vein along the superior border of the atrium. The striothalamic stem (4) and the internal cerebral vein (5) are also indicated.

(*B*) Corresponding roentgenogram with subependymal trunks numbered as in *A*.

vein of Rosenthal. Because of the relatively small size and deep sulci in the occipital and temporal lobes, the veins in these areas are shorter than elsewhere. The deep medullary veins in the parasagittal areas of the brain take an arcuate course convex laterally as they run downward. These veins also converge to the superior lateral corner of the lateral ventricle. At this point, they may drain into branches of either the medial or lateral group of subependymal veins. In contrast to the radiations of the corpus callosum, the body of the corpus callosum contains relatively few and short medullary veins which run either upward into the anterior and posterior cerebral veins or downward into subependymal roof veins of the lateral ventricle.

While this report is concerned primarily with the medullary veins, it must also be noted that there is a large number of medullary arteries arising from the arterial trunks on the surface of the brain. These vessels leave the surface arteries almost at right angles and promptly penetrate through the cortical gray matter and run for some distance in the white matter toward the ventricle. Despite their relatively small caliber, these arteries are unusually long with a surprisingly straight rectilinear course which may parallel the medullary veins for a considerable distance. In cases in which both medullary arteries and deep medullary veins are dilated, the appearance of these vessels may be quite similar. Differentiation must be based on recognition of the origin of the medullary arteries from arterial trunks on the surface of the brain and the termination of the medullary veins deeply in the subependymal veins.

Under normal conditions (Fig. 3 through 6, inclusive), medullary veins can rarely be clearly identified on carotid angiograms. Short portions near the surface of the brain and near the ventricle may be discerned on close observation of angiograms of good quality. From a theoretic point of view, it might be anticipated that the deep medullary veins might become sufficiently dilated to become easily visible in those pathologic conditions of the cerebrum associated with increased blood flow and increased venous drainage. These requirements are fulfilled by two well-known pathologic processes, arteriovenous malformations and highly vascular tumors, specifically glioblastoma

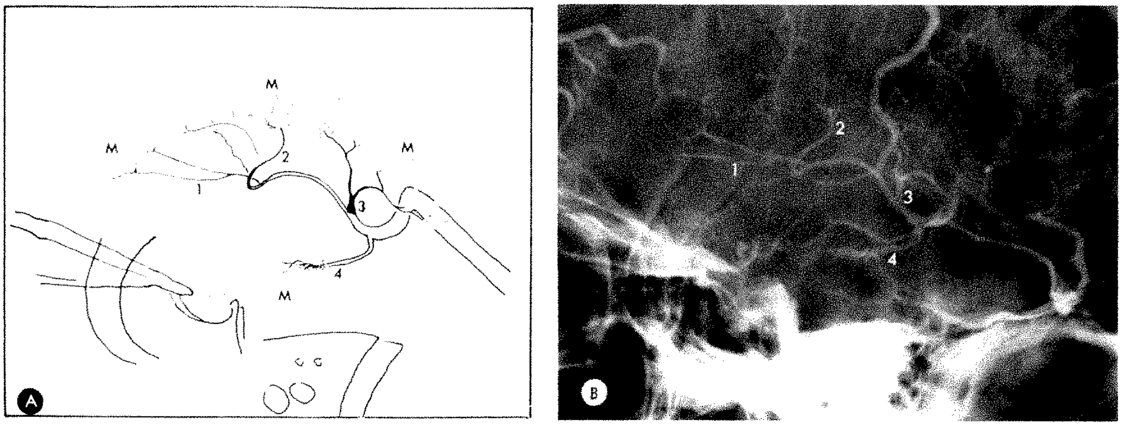

Fig. 4. Normal appearance.

(*A*) Tracing of lateral view. Numerous deep medullary veins (M) enter the vein of the septum pellucidum (1), the striothalamic stem (2) and an atrial subependymal vein (3). In addition, small anterior temporal deep medullary veins (M) enter the basal vein (4) via small temporal tip subependymal veins.

(*B*) Corresponding roentgenogram.

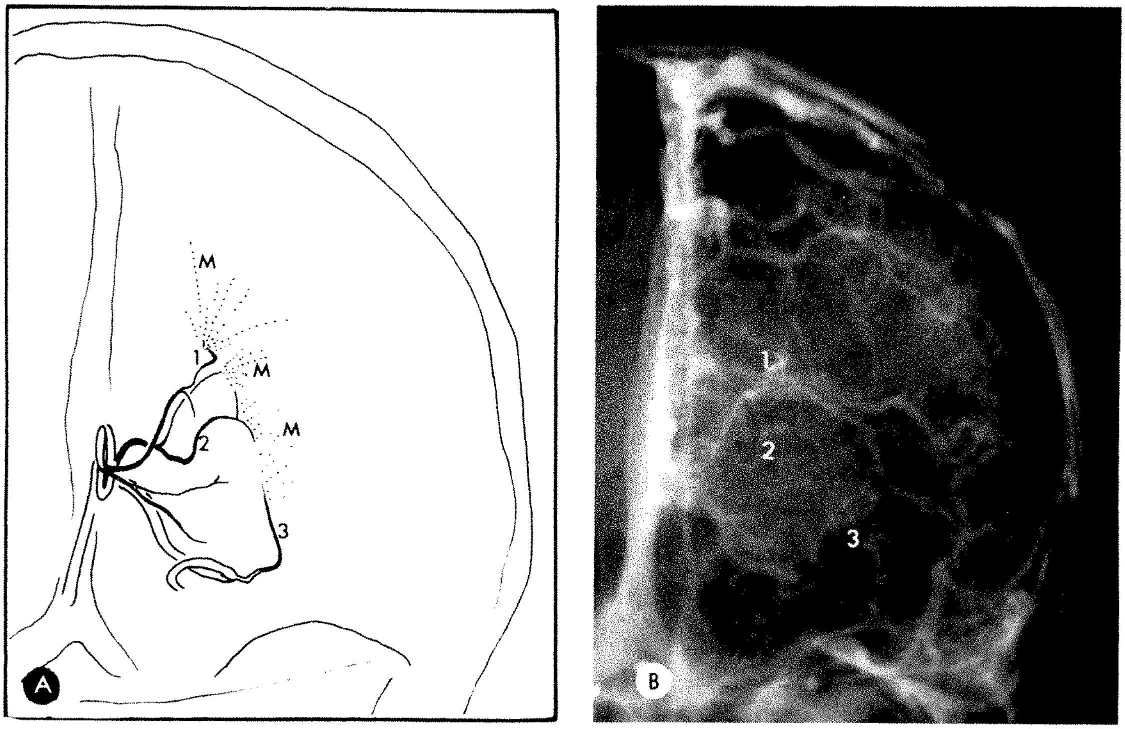

Fig. 5. Normal appearance.

(*A*) Tracing of anteroposterior view. A large number of veins—superficial cortical, subependymal, galenic and intracerebral—overlap one another in this projection. The smallest of these vessels can be identified as deep medullary veins (M) by their convergence and drainage into subependymal venous channels. Deep medullary veins join longitudinal caudate (1) and striothalamic veins, the direct lateral (2) and the inferior ventricular (3) subependymal veins.

(*B*) Corresponding roentgenogram.

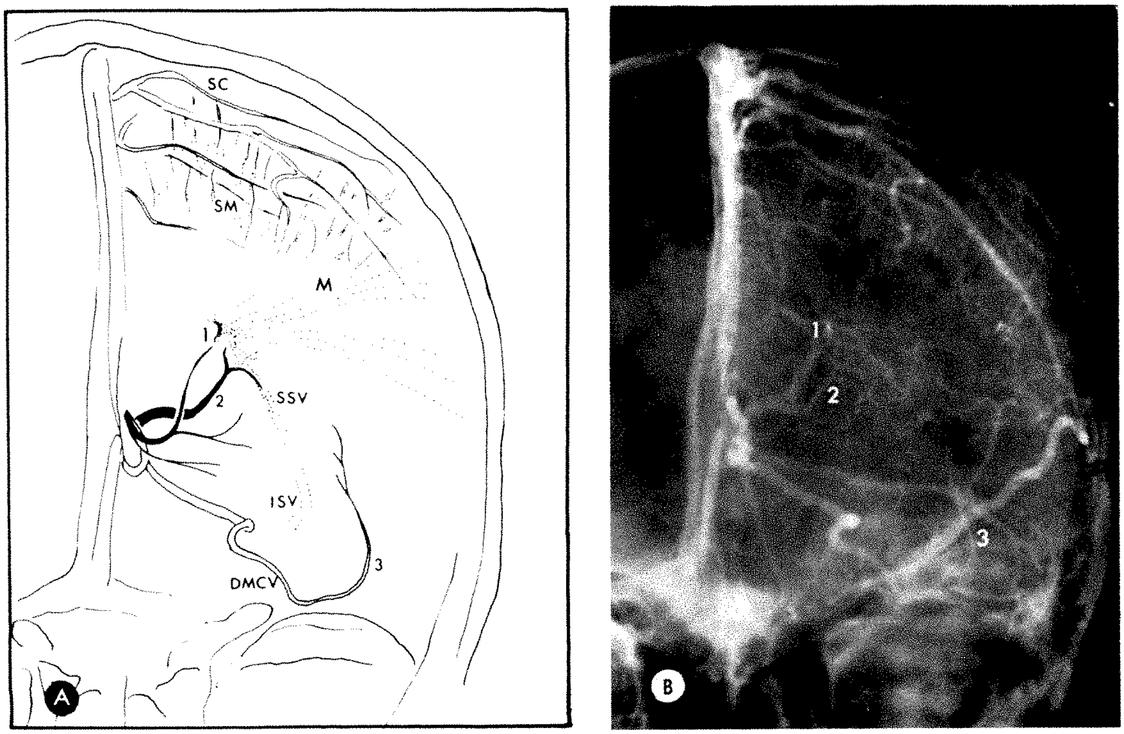

Fig. 6. Normal appearance.

(*A*) Tracing of anteroposterior view. A spray of deep medullary veins (M) traverses almost the entire thickness of the cerebral hemisphere to enter longitudinal caudate (1), striothalamic and direct lateral (2) subependymal veins. Superior striate veins (SSV) run upward to join longitudinal caudate veins. Portions of inferior striate veins (ISV) are also indicated. A large insular vein (3) continues directly into the deep middle cerebral vein (DMCV). Many short fine superficial medullary veins (SM) enter superficial cortical veins (SC) at a right angle. The boundary between superficial medullary and superficial cortical veins is rarely clear roentgenographically.

(*B*) Corresponding roentgenogram.

multiforme. While some arteriovenous malformations are confined to the surface of the brain with venous drainage exclusively via superficial medullary and cortical veins, the majority of such malformations are also associated with drainage via deep medullary veins into the subependymal vessels. If the arteriovenous communication is single or small, there may be only a few superficial and deep medullary veins participating in the process (Fig. 7, *A* and *B*). In those instances involving larger or multiple arteriovenous communications, a large part of the brain substance may be replaced by dilated and somewhat plexiform medullary vessels (Fig. 8, *A* and *B*). Most of these dilated vessels were apparently originally medullary veins. A lesion of this type often shows a wedge-shaped configuration with base at the cortex and apex at or near the corner of the lateral ventricle. This pattern is clearly related and similar to the normal distribution of the deep medullary veins. The draining subependymal veins are usually dilated and tortuous, sometimes to an extreme degree. Multiple superficial medullary veins also become dilated and tortuous but are ordinarily fewer in number and shorter than the deeper vessels.

The vascular architecture of glioblastoma multiforme is fairly well-known as a result of studies of pathologic specimens by several authors.[2,3,6,11] The central portions of such tumors frequently contain throm-

bosed vessels or vessels closed as a result of intimal proliferation. The diminution in blood flow in the central portions of glioblastoma multiforme is presumably the cause of the frequent necrosis in this region. On the other hand, the actively growing periphery shows markedly increased vascularity with numerous bizarre tortuous and dilated vessels and multiple arteriovenous communications. Large sinusoidal veins within the tumor may drain directly into subependymal veins or indirectly into these vessels via dilated medullary veins. An important feature of the vascular architecture[2,3] of glioblastomas is the fact that the increased vascularity is not confined to the actively growing peripheral portions of a glioblastoma multiforme but is also seen in the normal white matter adjacent to and surrounding the tumor. Russell *et al.*[10] described long thin-walle
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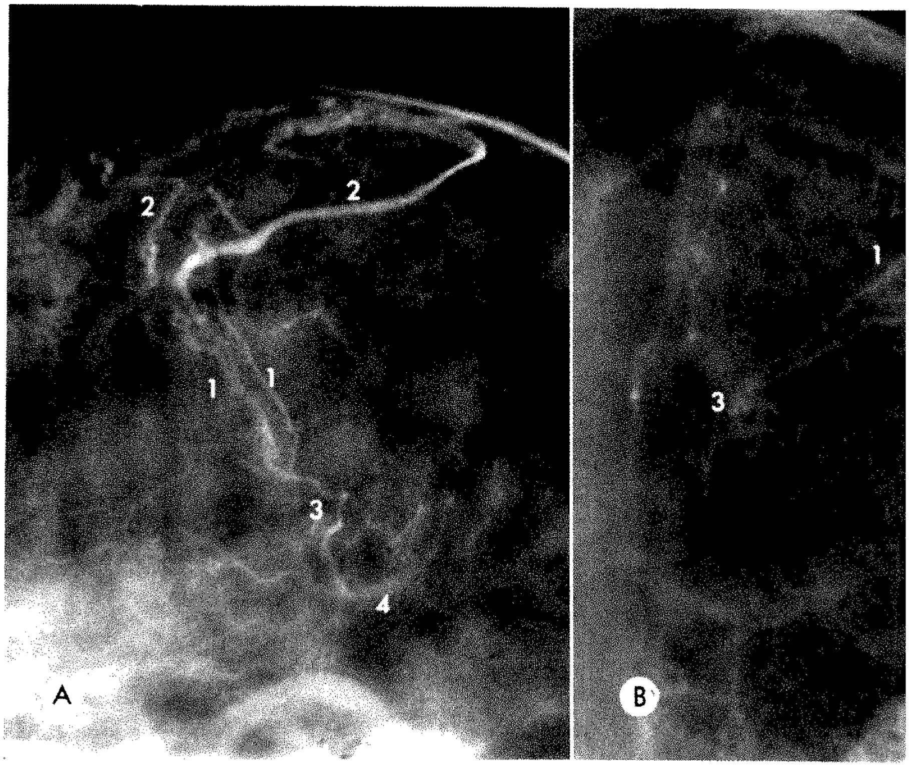


FIG. 7. Small arteriovenous malformation in the posterior frontal region.

(*A*) Lateral projection during the venous phase shows several prominent deep
which extend between superficial cortical veins (2) and the direct lateral subependym
vein enters the vein of Galen (4).

(*B*) Anteroposterior view shows long dilated medullary veins (1) extending from t
ependymal veins in the lateral wall of the ventricle (3).

ure 6. Continuing extracapsular
section.

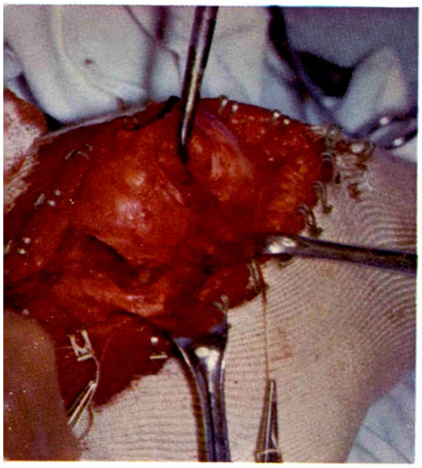
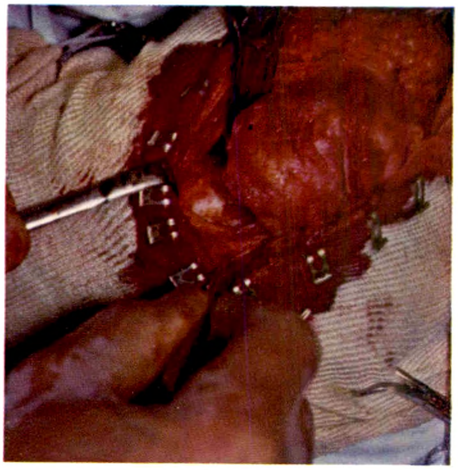
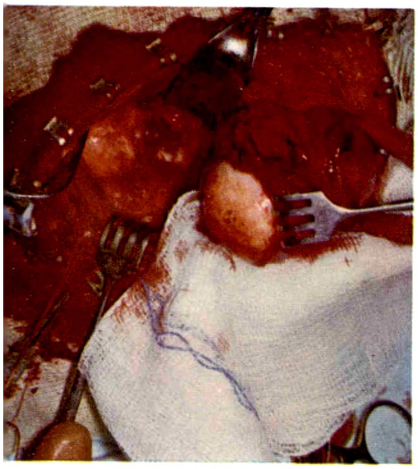


Figure 7. Continuing extracapsular dissection showing false acetabulum.





re 9. Beginning construction of new
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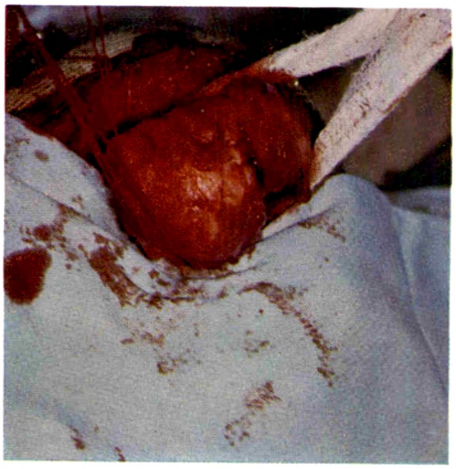
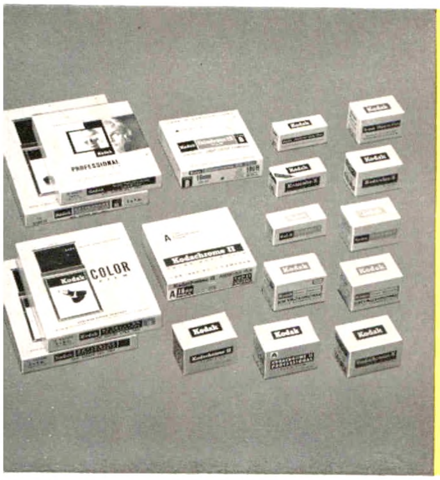


Figure 10. Closure of redundant capsule over femoral head.

Figure 11. Demo
acetabulum cente



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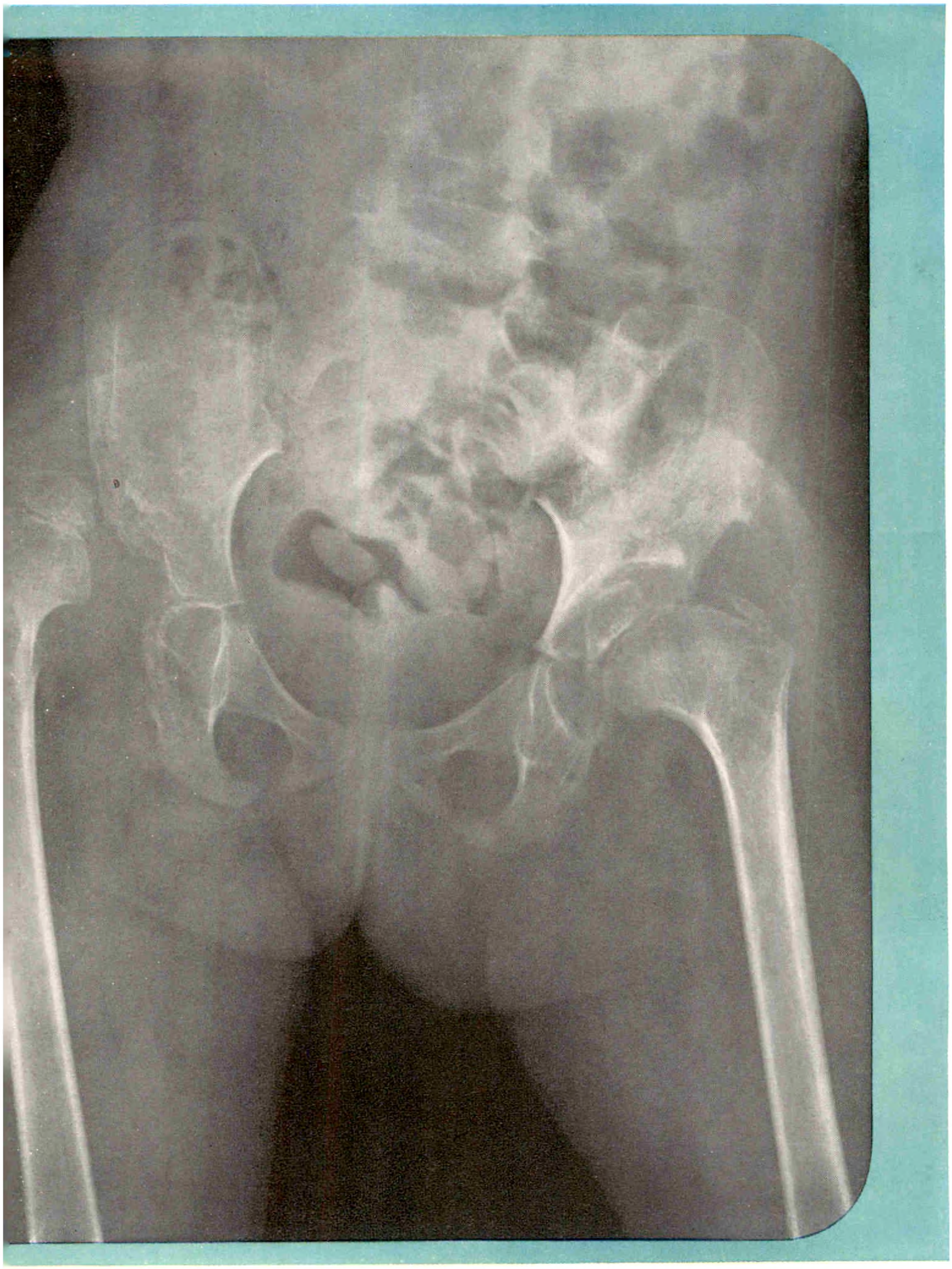

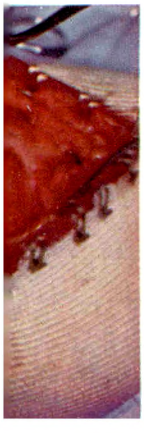

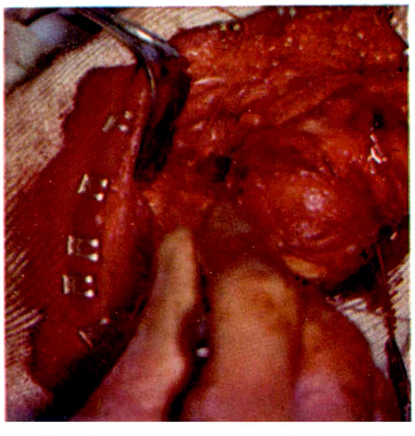

Figure 2. Operative field.

Figure 3. Posterolateral incision.

Figure 4. Exposure of greater trochanter.

Figure 5. Mobilization of attachments of abductors and external rotators.

For further details—
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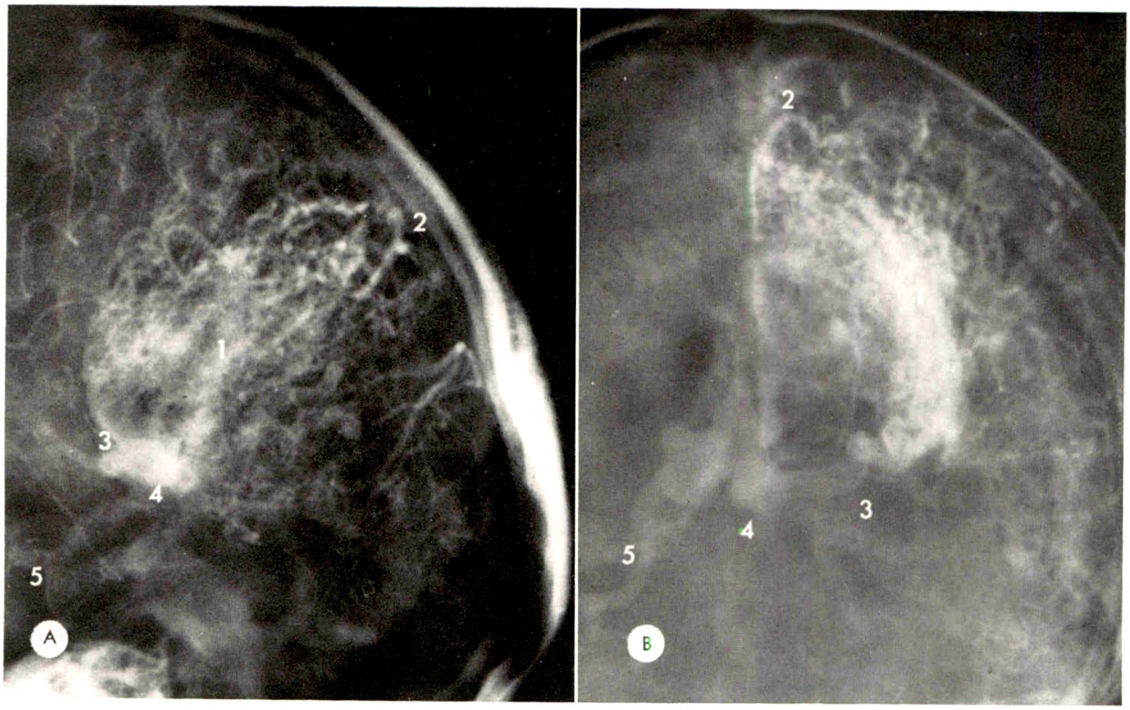

Fig. 8. Large arteriovenous malformation.

(*A*) The lateral projection shows innumerable large medullary veins (1) which drain partly superficially (2) but predominantly deeply (3). The deep drainage is into markedly dilated direct lateral subependymal veins and via these vessels into the internal cerebral vein (4). Two large basal veins (5) are faintly opacified.

(*B*) Anteroposterior projection shows that the large somewhat plexiform medullary veins (1) correspond in location and course to the parasagittal group of deep medullary veins. They run in a curved course from the medial surface of the hemisphere (2) into the huge direct lateral subependymal vein (3) and internal cerebral vein (4). The dilated basal vein on the right side (5) is fairly well opacified. The dilated basal vein on the left is less well opacified.

lated deep medullary veins in the periphery of and adjacent to the mass (Fig. 9 through 11, inclusive). The deep medullary veins under such circumstances have a characteristic appearance because they maintain their long relatively straight course and converging pattern. They are not markedly deviated presumably because of the infiltrating nature of the tumor and the poor demarcation between the tumor and adjacent brain. The typical pattern may, however, be difficult to discern unless a suitable projection is available. With tumors adjacent to the sylvian fissure, the medullary veins run more or less in the frontal plane. As a result, the anteroposterior projection will ordinarily show the typical wedge-shaped or converging vascular pattern to best advantage. With tumors in the frontal, occipital and temporal poles, the wedge-shaped pattern is easier to recognize in the lateral projection since the veins run closer to the sagittal plane (Fig. 11, *A* and *B*). The presence of relatively straight, crowded, converging veins produces a typical "streaming" or "spray" appearance, the apex of the stream or spray directed towards the ventricle. The venous pattern can usually be traced into one or several subependymal veins which occasionally are remarkably tortuous and dilated. The pattern of dilated medullary veins may not be seen until the capillary or early venous phases but is often evident earlier when arteriovenous anastomoses are prominent (Fig. 10, *A* and *B*; and 11, *A* and *B*). In fact, both medullary arteries and veins are sometimes simultaneously visualized and contribute to the streaming pattern.

From the point of view of localization, the appearance of the deep medullary veins in glioblastoma multiforme of the body of the corpus callosum is of particular interest (Fig. 12, *A*, *B*, *C* and *D*). In such instances, if only one side is injected, the appearance may be quite deceiving. In the anteroposterior projection, numerous dilated parasagittal deep medullary veins run in arcuate fashion toward the lateral corner of the anterior horn or the body of the ventricle. No similar vessels may be seen in the region of the corpus callosum or immediate supracallosal regions and the impression may be gained that the neoplasm is more laterally located. However, when the opposite side is injected, a surprisingly symmetric pattern of the parasagittal deep medullary veins is demonstrated with the region of the corpus callosum again appearing quite avascular. The reason for this central avascularity is not entirely clear but is presumably related to the relatively few normal medullary veins in this region and to the fact that the central portions of large glioblastomas often have relatively little blood flow.

In the majority of cases of glioblastoma multiforme of the cerebral hemispheres, there is also some venous drainage via superficial medullary veins. Because of the shortness of these vessels, they contribute relatively little to the streaming appearance of medullary veins described above. The superficial medullary veins may be

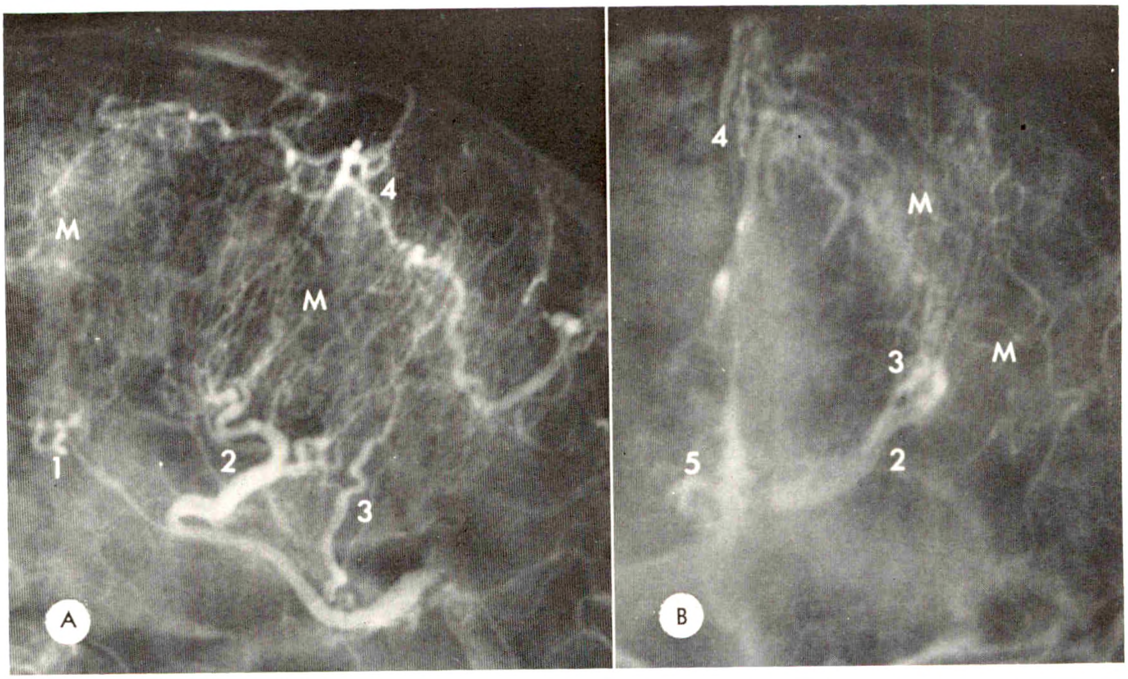

Fig. 9. Parasagittal glioblastoma multiforme.

(*A*) The lateral projection shows innumerable medullary veins (M) occupying the frontal and anterior parietal areas. Long, deep medullary veins run toward the ventricle in streaming fashion and join tortuous dilated subependymal veins—the vein of the septum pellucidum (1), the striothalamic trunk (2) and the direct lateral vein (3). There is a large transverse, tortuous, superficial cortical vein (4) into which numerous short, dilated, superficial medullary veins drain.

(*B*) Anteroposterior projection shows deep medullary veins (M) running to the superior lateral corner of the lateral ventricle and entering dilated striothalamic (2) and direct lateral (3) subependymal veins. A portion of the venous drainage is superiorly and medially into a superficial cortical vein (4). The internal cerebral vein (5) is displaced towards the opposite side.

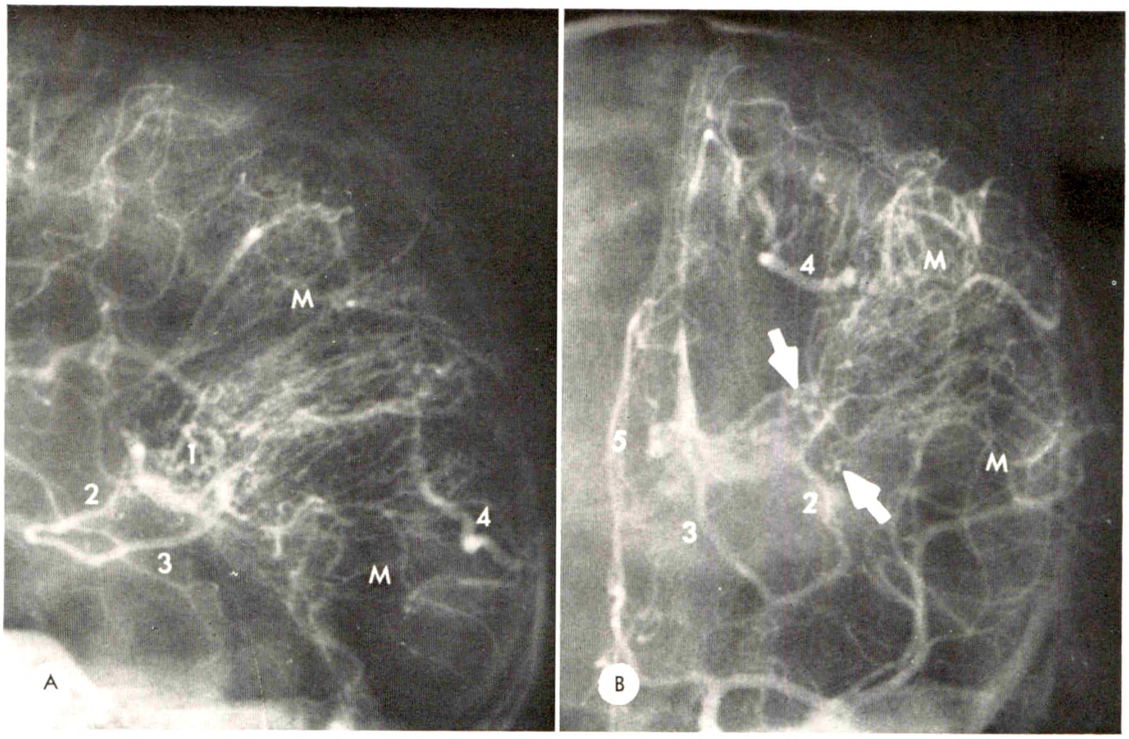

Fig. 10. Parieto-occipital glioblastoma multiforme.

(*A*) Innumerable closely crowded deep medullary veins (M) in the posterior parietal area stream downward and forward and converge to dilated tortuous subependymal veins in the region of the atrium (1). One of these subependymal veins, the lateral atrial subependymal vein (2), runs forward in the roof of the temporal horn and then enters the basal vein (3). A prominent superficial cortical vein (4) drains into the superior longitudinal sinus.

(*B*) Anteroposterior projection shows a wedge-shaped pattern of deep medullary veins (M) running downward and medially to join veins in the wall of the atrium (between arrows). Subependymal veins run medially to join the internal cerebral vein (5). The large lateral atrial subependymal vein (2) runs downward and forward in the lateral wall of the atrium and then leaves the temporal horn by turning medially to enter the basal vein (3). The draining superficial cortical vein (4) is again seen extending toward the superior longitudinal sinus. It should be noted that this is a late arterial phase and it is likely that the streaming medullary vessels consist not only of deep medullary veins but also of medullary arteries.

quite numerous but often appear fewer in number and individually larger and more tortuous than the deep medullary veins.

DISCUSSION

The recognition on angiography of arteriovenous malformations ordinarily presents no great problem. The venous components of these malformations described above are of interest primarily from the point of view of the origin and the treatment of such lesions. On the other hand, identification of deep medullary veins may be of considerable assistance in the diagnosis of a glioblastoma multiforme. In approximately 10 per cent of such cases, the characteristic sinusoidal dilatations and arteriovenous anastomoses have not been prominent while deep medullary veins have been obvious. Occasionally, as a result of necrosis or marked cyst formation, much of a large glioblastoma may appear to be avascular and the stain therefore quite atypical. The recognition of medullary veins in the periphery of such a lesion may permit the correct diagnosis (Fig. 13). In our experience to date, the streaming pattern of numerous dilated deep medullary veins has been seen in only one other type of tumor. This was a tumor involving the corpus callosum and adjacent portions of the frontal lobes which was reported as a perithelial sarcoma (Fig. 14). It is perhaps not surprising that a tumor of this type might show prominent medullary veins. We have had no further experience with tumors arising from blood vessels. We have seen no instance of oligodendroglioma, ependymoma or Grade I astrocytoma with this pattern presumably because of the relative scarcity of vessels in these tumors. Meningiomas do not show this pattern for the reason that drainage of a meningioma via deep medullary veins must be extremely rare if it occurs at all. In an occasional case of glioblastoma, the scalp or meningeal vessels may appear large and the stain sufficiently atypical to suggest the possibility that the lesion may be a meningioma. In such instances, visualization of deep medullary veins excludes this diagnosis (Fig. 15, *A* and *B*). It should be noted that with a meningioma, dilated superficial cortical veins may be displaced so deeply as to simulate vessels within the brain substance (Fig. 16, *A* and *B*). It is evident, however, in such instances that these vessels do not correspond in configuration or course to deep medullary veins and that they drain into superficial cortical rather than subependymal veins.

The complete picture of numerous streaming deep medullary veins described above has been quite a reliable sign of glioblastoma multiforme. There are, however, cases with fewer enlarged deep medullary veins which present a different pattern. The background of innumerable fine undisplaced vessels is not present. Relatively few but markedly enlarged medullary veins often with branching tributaries appear to be displaced in arcuate fashion around the periphery of the tumor, approach each other near the ventricle and join subependymal trunks directly. This pattern apparently can be produced not only by a glioblastoma (Fig. 17) but also by an astrocytoma Grade II with central necrosis (Fig.

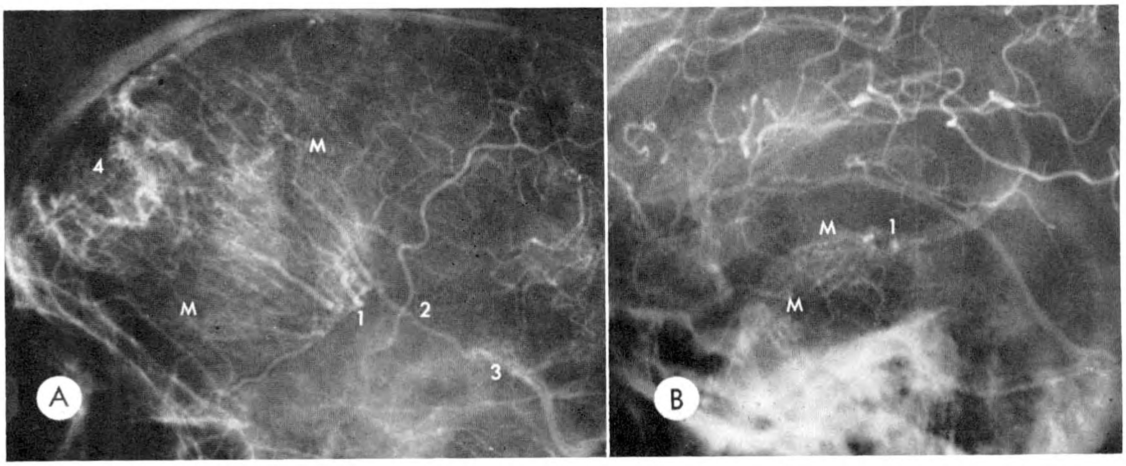

FIG. 11. Two cases of glioblastoma multiforme.

(*A*) In this patient, the lateral projection shows innumerable streaming deep medullary veins (M) which run downward and backward to join a dilated longitudinal caudate vein (1) in the anterolateral corner of the frontal horn. Further drainage of these vessels is into the dilated septal vein (2) and then into the internal cerebral vein (3). The sharp demarcation of the deep medullary veins along the anterior margin of the frontal horn (1) indicates that this wall is flattened and displaced backward. There are also several tortuous cortical veins (4) over the frontal pole.

(*B*) Glioblastoma multiforme located in the anterior portion of the temporal lobe. A large number of deep medullary veins (M) in the anterior portion of the temporal lobe run backward and upward to converge on the temporal horn. These vessels join subependymal veins of the temporal horn and drain into the basal vein (1).

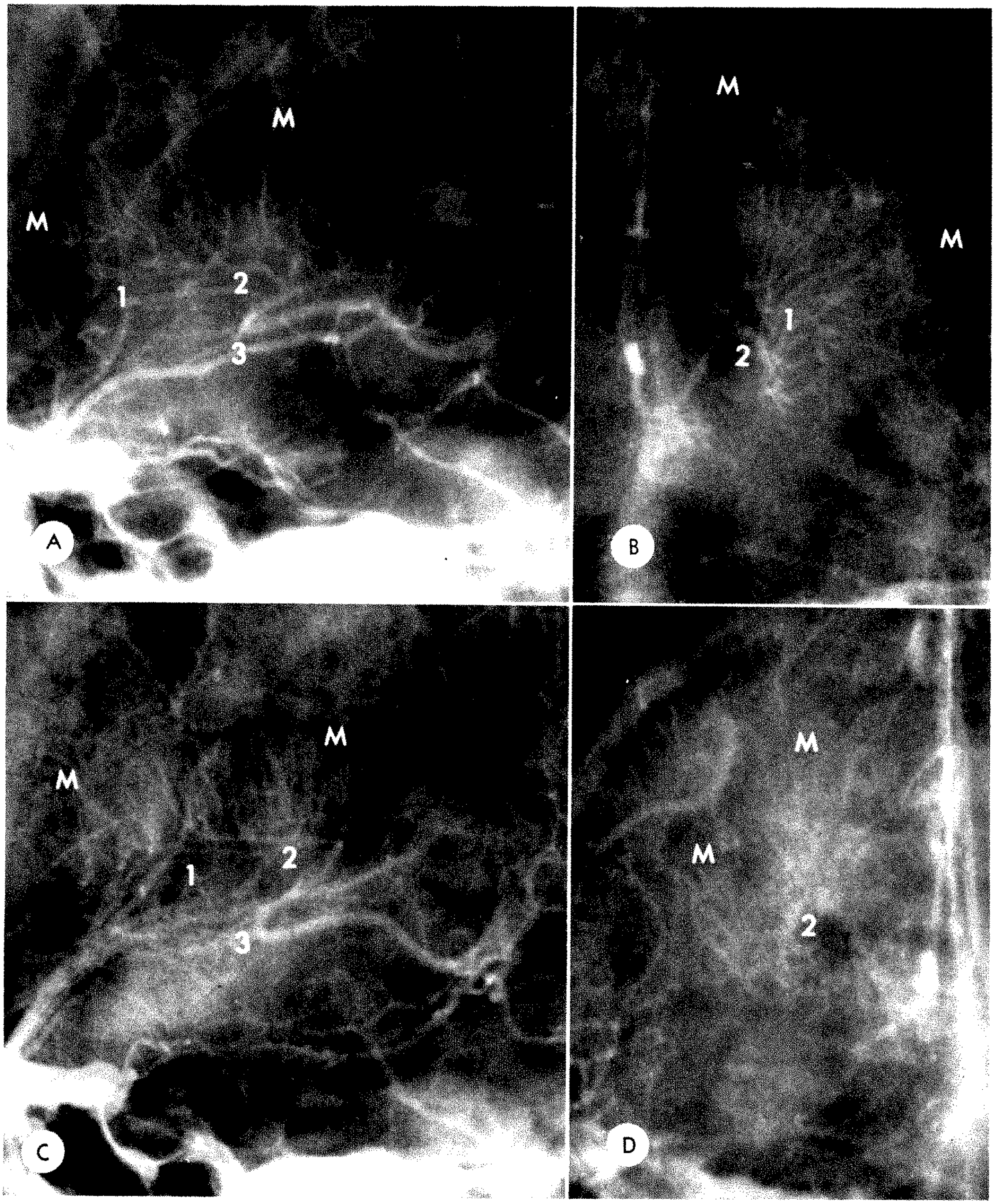

FIG. 12. Glioblastoma multiforme of the corpus callosum. Bilateral carotid angiography.

(*A*) Left sided injection. The lateral projection shows dilated medullary veins (M) in the posterior frontal region which drain downward into the longitudinal caudate (1) and anterior caudate (2) veins. The subependymal veins as well as the anterior end of the internal cerebral vein (3) are displaced downward.

(*B*) Injection on the same side, anteroposterior projection. The dilated medullary veins (M) are located in the parasagittal and suprasylvian areas. These vessels stream in fan-like fashion to converge on the longitudinal caudate (1) and anterior caudate veins (2). The internal cerebral vein is not displaced from the mid-line. No dilated superficial cortical veins are evident.

(*C*) Right sided injection. The lateral projection shows an identical appearance of the medullary (M) and subependymal (1, 2, 3) veins to that seen on the opposite side.

(*D*) Right sided injection. The anteroposterior projection shows poorer filling than on the left side but the findings are identical.

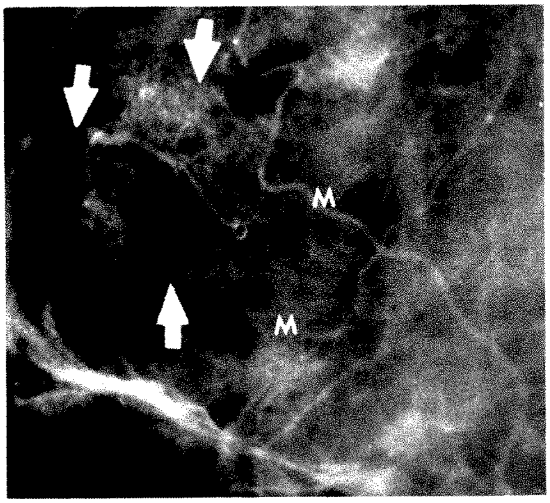

FIG. 13. Huge cystic glioblastoma multiforme. A rather discrete circular "stain" is seen in the region of the frontal pole (between arrows). Behind this, a small wedge-shaped collection of deep medullary veins (M) is present converging backward toward the frontal horn. Despite the fact that the number of medullary veins evident is not large, the diagnosis of glioblastoma multiforme was suggested. At operation, this was confirmed. Most of the tumor consisted of a huge cyst.

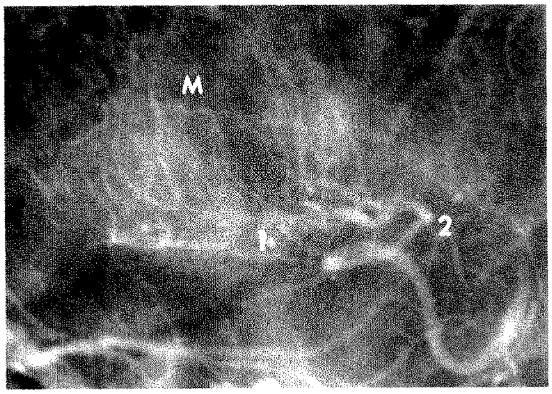

FIG. 14. Perithelial sarcoma involving the corpus callosum and adjacent portions of the frontal lobes. The lateral projection shows a large number of dilated medullary veins (M) streaming downward and slightly backward to join the longitudinal and anterior caudate vein and the striothalamic stem (2). Medullary veins also enter the dilated septal vein (1). The dilated longitudinal caudate vein is displaced downward and flattened.

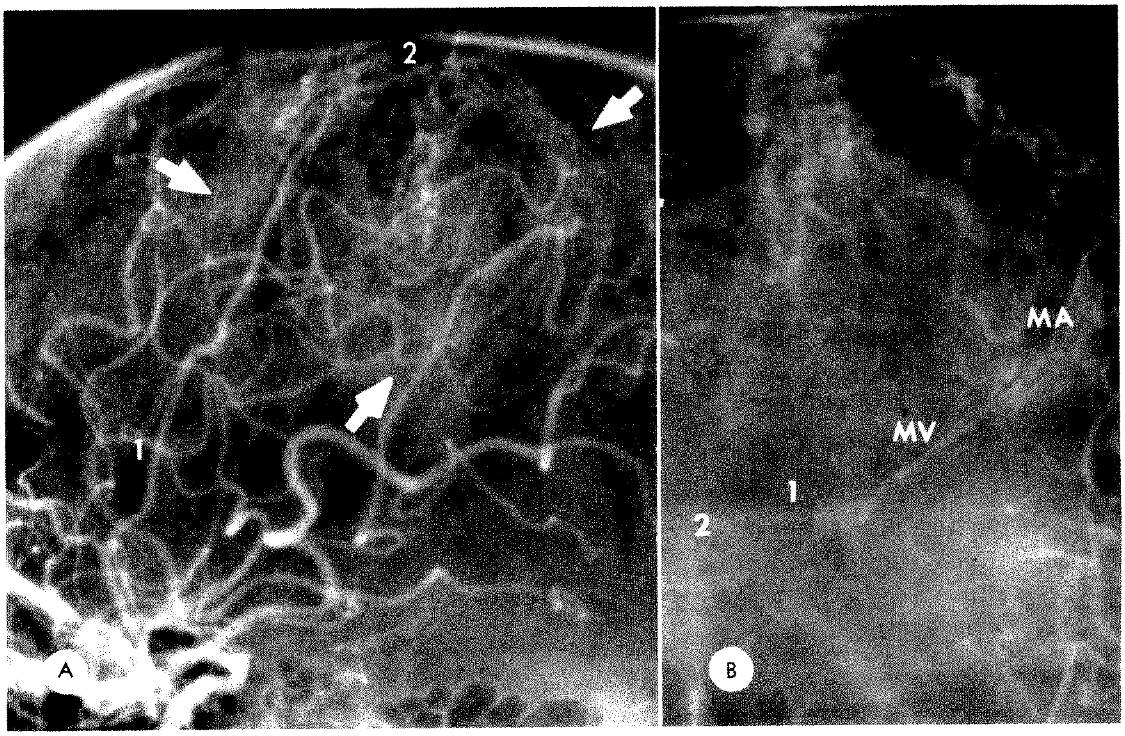

FIG. 15. Differential diagnosis, glioblastoma multiforme or meningioma.

(*A*) Arterial phase in lateral projection shows a stain (between arrows) in the posterior frontal and Rolandic areas. A large superficial temporal artery (1) extends to the vault of the skull (2) and seems to participate in the formation of the stain. This appearance suggests the possibility of a meningioma.

(*B*) Late arterial phase in the anteroposterior projection shows dilated medullary arteries (MA) which converge to prominent medullary veins (MV). These veins enter a stretched direct lateral subependymal vein (1) which drains into the displaced internal cerebral vein (2). This appearance contradicted the diagnosis of an extracerebral neoplasm and suggested the possibility of a glioblastoma multiforme. This was confirmed at operation.

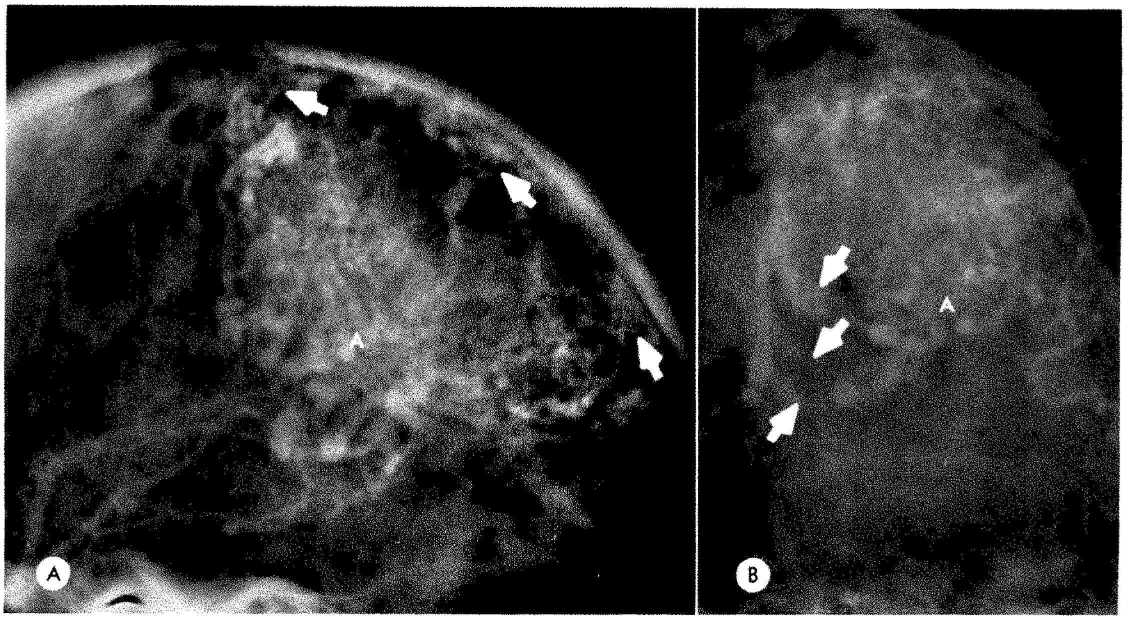

FIG. 16. Large parietal parasagittal meningioma.

(*A*) Lateral projection shows numerous markedly dilated tortuous veins (A) in the parietal region. Between these vessels and the calvarium there is a semilunar area which is relatively avascular and shows a faint homogeneous stain. Through this stain, several tortuous, superficial cortical veins (arrows) can be seen running superiorly.

(*B*) Anteroposterior projection shows the bizarre, tortuous, dilated veins (A) to be situated at a considerable distance from the inner table. Between these vessels and the inner table, there is a homogeneous faint stain without dilated vessels within it. The superficial cortical veins (arrows) evident in the lateral projection are seen to be located on the medial aspect of the hemisphere. Note that there is no apparent drainage via subependymal or deep veins, *i.e.*, the abnormal veins must be superficial cortical veins which are displaced markedly inward. The homogeneous stain between these veins and the inner table is typical of a meningioma.

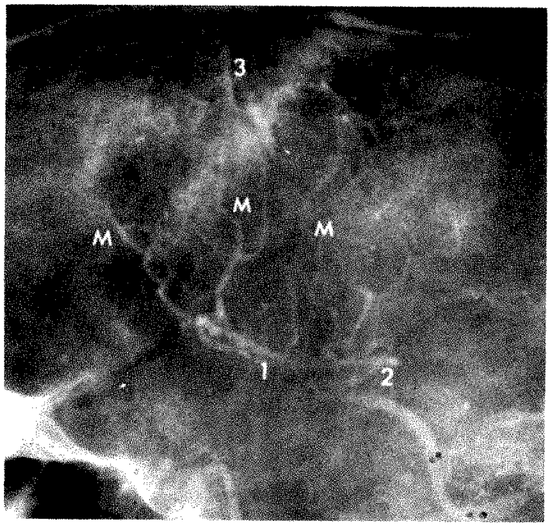

FIG. 17. Glioblastoma multiforme in the posterior frontal region. Several dilated deep medullary veins (M) enter the enlarged longitudinal caudate and anterior caudate veins (1) which drain into the striothalamic stem (2). The superior portion of the tumor is drained by a superficial cortical vein (3). In this patient, the more characteristic streaming appearance of innumerable deep medullary veins is not evident.

18, *A* and *B*). It should be emphasized that the complete streaming pattern occurs in only a minority of cases of glioblastoma and that the recognition of a few nondescript medullary veins is not likely to be of differential diagnostic importance. Moreover, failure to visualize deep medullary veins in any particular case does not exclude the possibility of a glioblastoma.

It is of interest that the streaming pattern of deep medullary veins draining a neoplasm has not been seen in instances of metastatic tumors despite the fact that many of these tumors are obviously highly vascular, for example, metastatic hypernephroma. This may be somewhat of a coincidence and related to the fact that the large majority of such metastatic tumors occur in the immediate subcortical portion of the cerebral hemispheres and are therefore drained by the superficial vessels. It is possible that a metastatic lesion in the wall of the ventricle might show drainage via subependymal veins. It is also reasonable to expect that intraventricular neoplasms of any type may show substantial drainage via subependymal veins. However, it is not likely that the streaming pattern of abnormal deep medullary veins will be seen in such cases since no substantial amount of white matter would be involved.

SUMMARY

1. The veins of the white matter of the cerebral hemispheres can be divided into superficial and deep medullary veins. Transitional or complete types such as anastomotic or transcerebral veins are also described.
2. The deep medullary veins show a very

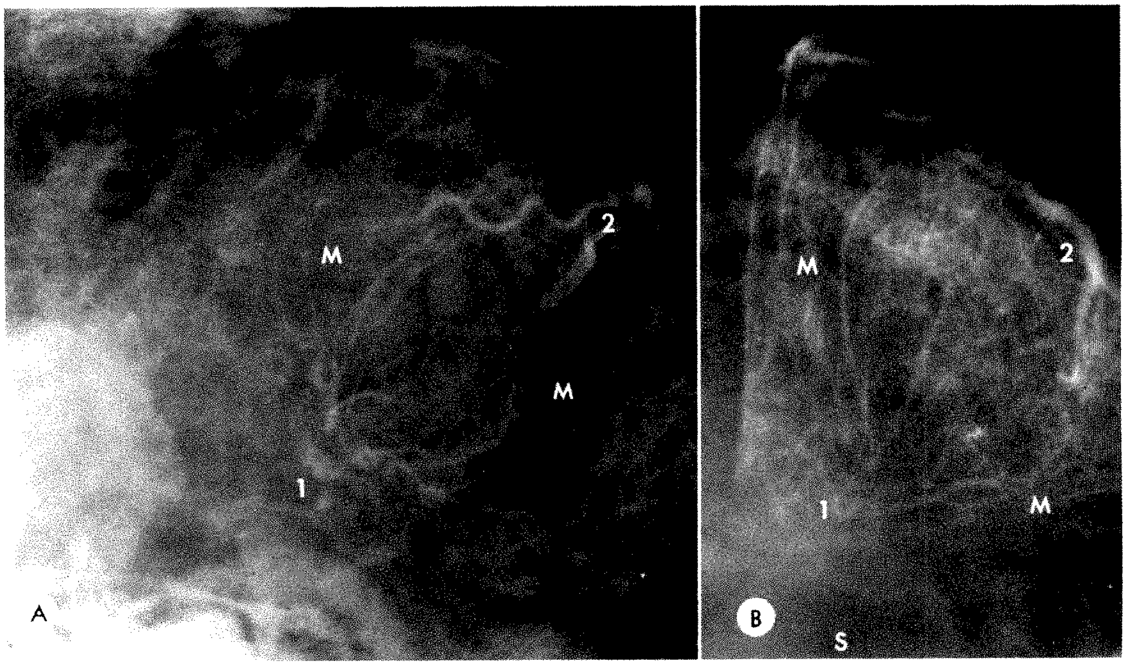

Fig. 18. Partially necrotic astroblastoma in a patient with a large sphenoidal ridge meningioma.

(*A*) Lateral projection. A large homogeneous stain (S) typical of meningioma is seen along the sphenoidal ridge. In addition, however, several large deep medullary veins (M) are seen in the parietal region which converge to subependymal veins in the atrium and appear to be displaced in an arcuate fashion around the periphery of a mass. There is also a prominent cortical vein (2) on the surface.

(*B*) Anteroposterior projection shows a similar appearance of several large widely separated deep medullary veins (M) converging to the corner of the lateral ventricle (1). The prominent superficial cortical vein (2) is again seen. The homogeneous stain due to the meningioma (S) is not clearly evident on this roentgenogram.

characteristic appearance and course as they converge toward specific portions of the lateral ventricle to drain into the subependymal veins.

3. The areas of convergence of the deep medullary veins to the wall of the ventricle appear to be related to the regions in which the subependymal gray matter persists in the adult.

4. Under normal circumstances, very few medullary veins are evident on angiography. However, their typical appearance may become evident as a result of dilatation associated with arteriovenous malformations or glioblastoma multiforme.

5. In some instances of glioblastoma multiforme, the recognition of a characteristic streaming pattern of numerous dilated medullary veins permits a reliable differential diagnosis. No similar pattern has been seen as yet in well-differentiated primary tumors of the cerebral hemispheres, in meningiomas, or in metastatic tumors.

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ANEURYSM OF THE VEIN OF GALEN*

CASE REPORT AND REVIEW OF THE LITERATURE

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ANEURYSMAL dilatation of the vein of Galen is a rare and interesting condition. The diagnosis is established primarily by the characteristic roentgenographic findings.[20] A case of a 4½ year old girl is presented in whom the diagnosis was made on plain roentgenograms and confirmed by arteriograms.

REPORT OF A CASE

This 4½ year old white girl was apparently well until the age of 3½ years when she fainted while playing. Dilated pupils, cyanosis, and repeated sneezing accompanied this attack. The results of the physical examination were unremarkable. Three months before admission she experienced two episodes of falling to the knees, without loss of consciousness. At those times the eyes rolled back and there was perioral cyanosis, followed again by repeated sneezing. The physical findings again were unmarkable, but roentgenograms of the skull revealed that a curvilinear calcification was present in the midline posteriorly (Fig. 1, *A* and *B*). Aneurysm of the vein of Galen was suggested on the basis of these roentgenograms. Cardiomegaly and increased pulmonary vascularity were noted on roentgenograms of the chest. The child was referred to the pediatric clinic where slight prominence of the superficial veins of the skin about the eye and bridge of the nose was noted (Fig. 2). A blowing, high-pitched murmur was heard on auscultation of the skull over the parietal region. Arteriograms were obtained, using a Sanchez-Perez Rapid Film Changer.* Bilateral carotid arteriograms showed no filling of the aneurysm but there was retrograde filling of a dilated right vertebral artery through an anomalous anastomosis of the left pericallosal artery (Fig. 3, *A* and *B*) with the right posterior cerebral artery. Venous drainage was through multiple superficial anomalous channels (Fig. 4). Left vertebral arteriography showed the aneurysm to be filled through enlarged vertebral and posterior cerebral arteries (Fig. 5, *A* and *B*; and 6, *A* and *B*).

Table I

ANEURYSM OF VEIN OF GALEN IN 37 PATIENTS REPORTED IN THE LITERATURE

Sex	
Males	23
Females	10
Unknown	4
Age Incidence	
Newborn	11
1–6 mo.	4
7–12 mo.	5
13 mo.–2 yr.	5
3–5 yr.	3
6–10 yr.	0
11–20 yr.	3
21–30 yr.	2
31–40 yr.	1
41–50 yr.	1
More than 50 yr.	0
Unknown	2
Presenting Signs and Symptoms	
Prominent superficial veins	13
Respiratory distress	11
Cranial bruit	9
Cardiomegaly or other signs of congestive failure	9
Craniomegaly	8
Cyanotic episodes	8
Convulsions	7
Arteries Supplying Aneurysm (29 patients)	
Posterior cerebral	23
Superior cerebellar	3
Vertebral	3
Anterior choroidal	3
Basilar	3
Pericallosal	3
Middle cerebral	1

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* Automatic Seriograph Corp. (subsidiary of Litton Ind.), College Park, Maryland.

* From the Department of Radiology, University of California School of Medicine, San Francisco, California.

DISCUSSION

Since first mentioned by Dandy[5] in 1928, a total of 37 cases of aneurysm of the vein of Galen have been reported in the world literature.[1-23] In the present report, an attempt has been made to exclude patients in whom the vein of Galen was only slightly dilated in association with a large arteriovenous malformation.[11] Most of the aneurysms were diagnosed in the immediate postnatal period or before the patient's sixth year, although one was diagnosed in a 49 year old patient[19] (Table I). Twice as many males as females are affected. Respiratory distress and cyanosis are the most common presenting symptoms, with enlarging head size, convulsions, and various disorders of the central nervous system reported in decreasing frequency. The most common physical findings include prominent superficial veins of the face and scalp, cranial bruit, cardiomegaly, and signs of congestive heart failure.

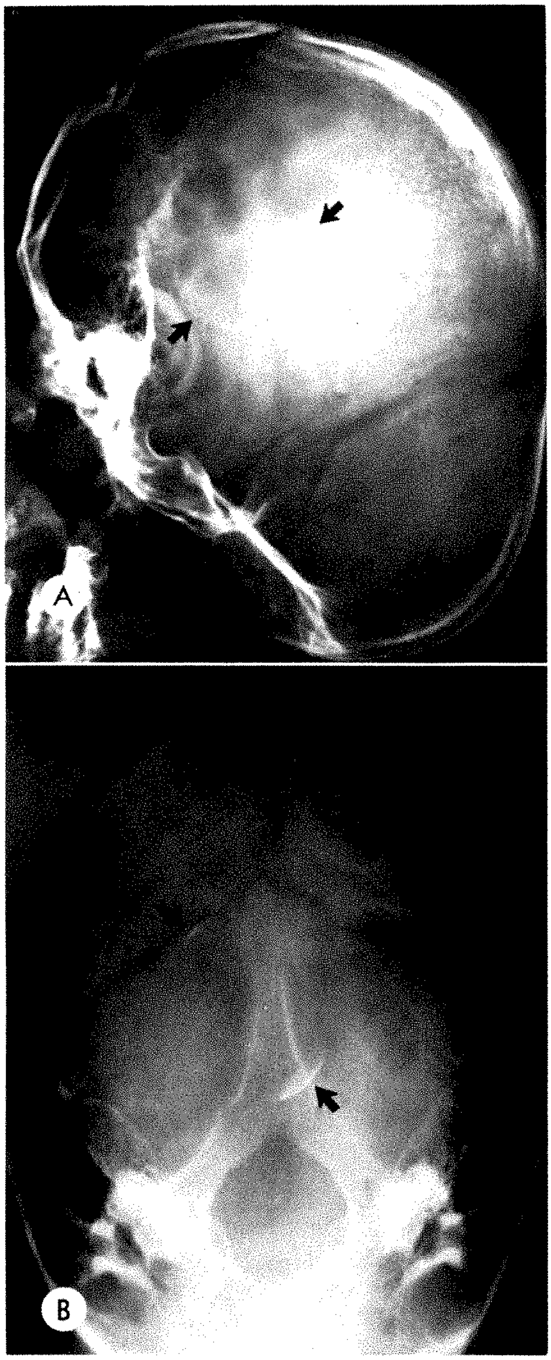

Fig. 1. (*A* and *B*) Large curvilinear calcification in the region of the vein of Galen.

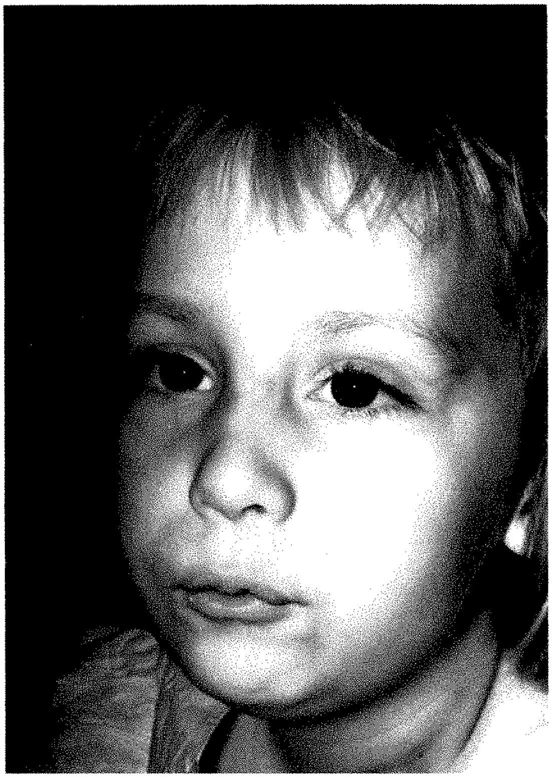

Fig. 2. Infrared photograph. Prominent superficial veins about the nose and left eye.

Plain roentgenograms of the skull were mentioned in 19 of the patients, 7 demonstrating a characteristic curvilinear calcification in the region of the vein of Galen. In none of the previously reported patients

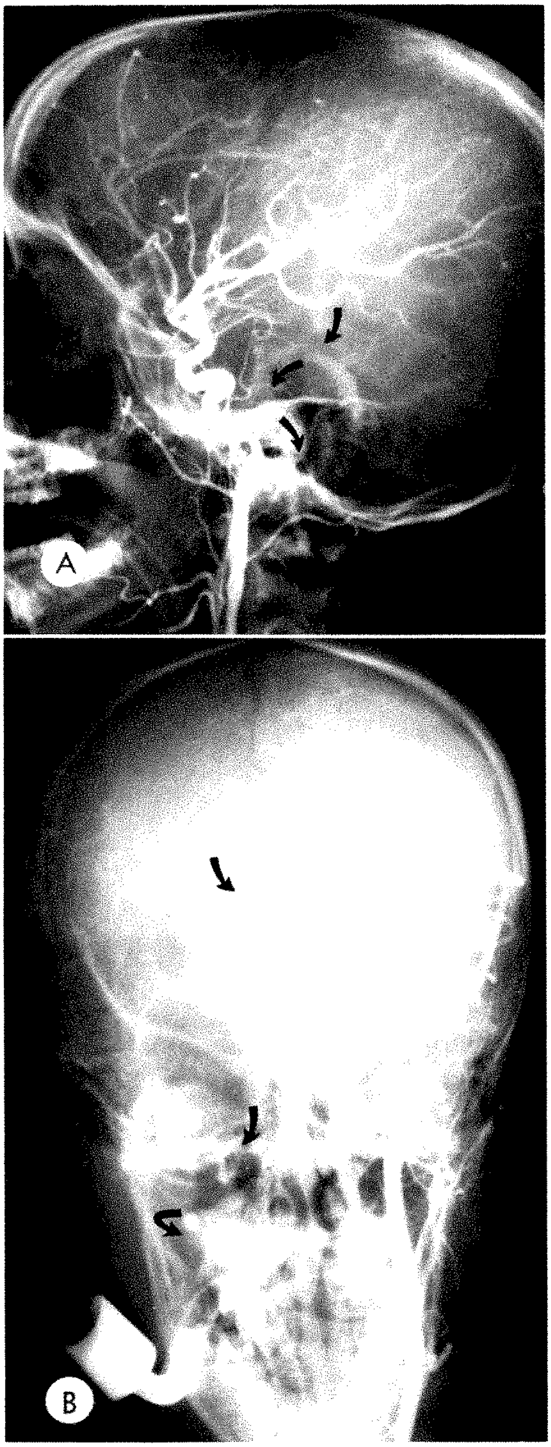

Fig. 3. Left carotid arteriogram. (*A* and *B*) Retrograde filling of the right vertebral artery through an anomalous anastomosis between the pericallosal and posterior cerebral arteries. The aneurysm of the vein of Galen is not opacified.

was calcification noted before the age of 15 years. Ten gave evidence of craniomegaly, and only 2 were reported as normal. Pneumoencephalography or ventriculography was performed in only 9, resulting in evidence of hydrocephalus in 7 and no abnormality in 2.

Angiograms were obtained on 17 of 37 patients. Eleven of these 37 were alive at the time of the reports (6 improved or asymptomatic and 1 "retarded" after operation; 4 untreated). The state of one patient was unknown. Necropsy findings were available on the others. Information from necropsy or arteriography regarding the arterial supply to the aneurysm was mentioned in 29 of the 37 patients (Table 1). The posterior cerebral artery is the most common feeding vessel. The superior, cerebellar, vertebral, pericallosal, and choroidal arteries are only occasionally involved. The venous drainage, when mentioned, was always via the straight sinus.

The present patient is of particular interest because of the calcification that was noted at the age of $4\frac{1}{2}$ years. Calcification of an aneurysm of the vein of Galen at this early age had not been reported previously. In contrast to the angiographic findings in reported cases, the straight sinus in this pa-

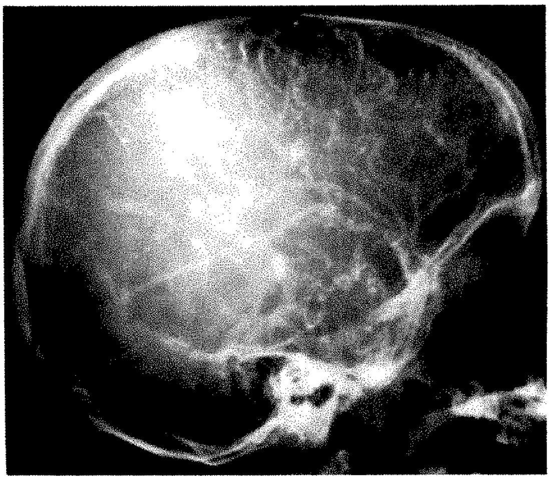

Fig. 4. Left carotid arteriogram, venous phase. Filling of numerous anomalous venous channels. Internal cerebral vein and straight sinus not opacified.

tient was not filled and was, presumably, occluded or compressed. This resulted in a venous return via circuitous superficial channels. The relatively small volume shunt may be the result of increased resist-

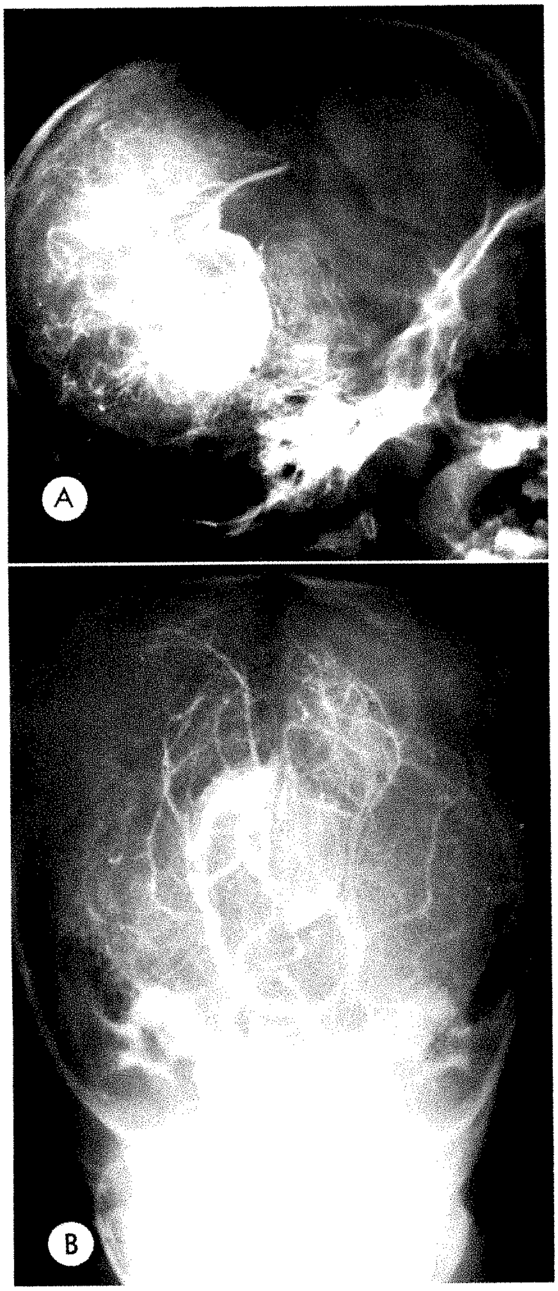

Fig. 5. Left vertebral arteriogram. (*A* and *B*) Enlargement of the vertebral and both posterior cerebral arteries. The aneurysm is filled primarily from the right posterior cerebral artery and possibly to a lesser degree from the left posterior cerebral artery. Retrograde filling of the pericallosal artery from an anomalous connection with the posterior cerebral artery.

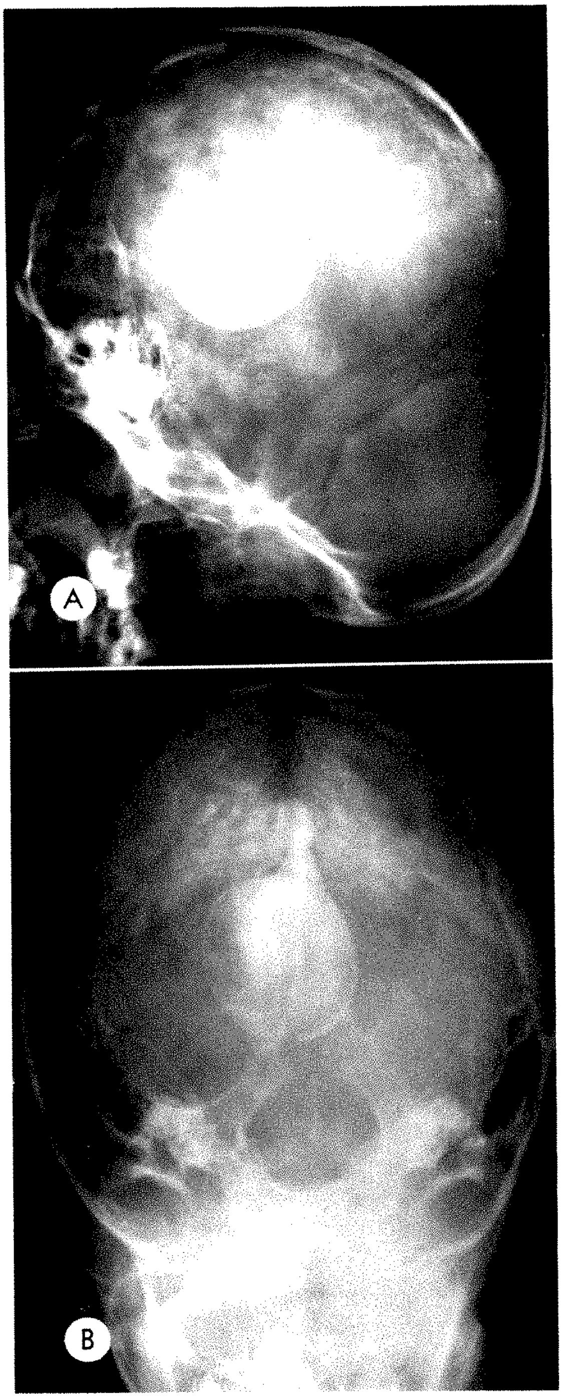

Fig. 6. Later phase of vertebral arteriogram. (*A* and *B*) Dense opacification of the aneurysm. Straight sinus not opacified.

ance in these multiple small collateral channels.

SUMMARY

Angiographic findings in a $4\frac{1}{2}$ year old girl with an aneurysm of the vein of Galen are reported. Calcification of the aneurysm and apparent occlusion of the straight sinus were two unusual features noted in a patient of this age.

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THE PRIMITIVE TRIGEMINAL ARTERY AS SEEN ANGIOGRAPHICALLY AND AT POSTMORTEM EXAMINATION*

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THE primitive trigeminal artery is an anomaly caused by the persistence of a presegmental dorsal artery accompanying the first branch of the trigeminal nerve. It represents one of the three well known and described carotid-basilar anastomoses, depending on their relation with the fifth, eighth and twelfth cranial nerves, and appears as a direct connection of the internal carotid and basilar arteries proximal to the siphon.

The embryologic development has been discussed by Padget,[43] De Vriese,[11] Örtel,[42] Hochstetter,[23] Altmann,[1] and other authors.

Prior to the era of cerebral angiography, only a few cases were reported as findings at autopsy. The persistence of this artery into adult life was first described by Quain[47] in 1844. Örtel,[42] in 1922, found 2 primitive trigeminal arteries in one specimen. The topographic relationship to the sella turcica, trigeminal nerve and other neighboring structures was dealt with in detail and illustrated by Sunderland[62] in 1948. Hasenjäger[21] and Ökrös,[41] in 1937, were the first to mention angiography as an aid in demonstrating this anomaly during life. However, no use was made of the procedure until 1950, when Sutton[63] recognized this anastomosis on angiograms, due to simultaneous filling of the distal basilar artery and posterior cerebral arteries following common carotid arterial injection. Since then many investigators have reviewed their series of angiographic studies and have reported such anastomoses.

The purpose of the authors is: (1) to present a chronologic review of the literature pertaining to the primitive trigeminal artery; (2) to describe the configuration of a primitive trigeminal artery as revealed by carotid angiography; and (3) to report 2 cases of carotid-basilar anastomosis, in 1 of which postmortem angiographic studies were performed.

The literature has been reviewed from 1844 to 1963 and the collected data are given in Table I, by listing the names of the authors with the reference numbers, the number of cases reported by each author, the sex, whether the case was diagnosed by angiography or at autopsy, the relative incidence to total examinations where this figure was available, and the side on which the anomaly was located.

Including the 2 cases described herein, a total of 134 cases of primitive trigeminal artery has been reported. One hundred and five of these were diagnosed by angiography and 38 at autopsy. Only 9 of the angiographically diagnosed cases were verified at autopsy. The sex incidence was found to be about equal with no significant preference for the right or left side. Most of these unusual anastomoses were incidental findings, not related to the patients' clinical symptoms and diagnosis. We found this to be true in our cases.

REPORT OF CASES

Case I (Hosp. # 210 898, C.M.). This 62 year old white female was a chronic alcoholic with previous major abdominal surgery for subtotal gastric resection and splenoportal shunt. From studies made to exclude an intracranial lesion, a right primitive trigeminal artery was diagnosed. During direct percutaneous common carotid artery injection, an immediate filling of the posterior circulation through a large anas-

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TABLE I

Year Reported	Author	No. of Cases	Sex		Diagnosis by Angiography (no. of cases /total no.)	Diagnosis at Autopsy (no. of cases /total no.)	Side	
			F	M			R	L
1844	Quain[47]	1				1		1
1855	Hochstetter[23]	2		1		2	1	1
1860	Tüngel[68]	1				1	1	
1862	Flesch[14]	1				1		1
1866	Decker[9]	1	1			1		
1874	Duret[12]	2				2	2	
1880	Tareniecki[64]	1				1	1	
1907	Blackburn[4]	1		1		1/220		1
1909	Smith[59]	1		1		1		1
1922	Örtel[42]	1	1			1	1	
1931	Kotrnetz[30]	4				4	3	1
1935	Feller[13]	1				1		
1936	Ökrös[41]	1				1		
1937	Hasenjäger[21]	1				1		
1938	Slany[58]	1	1			1	1	
1941	Hultquist[26]	1				1		
1941	Sunderland[61]	3		1		3/210	1	2
1950	Sutton[63]	2		2	1/1,000	1	1	1
1950	Lindgren[34]	2			2/1,526			
1952	Frugoni[16]	1			1			
1952	Philippides *et al.*[45]	1			1			
1953	Harrison and Luttrel[20]	3	1	2	3/582	1	2	1
1953	Stenvers *et al.*[60]	1		1	1/750			1
1953	Kloos[29]	1	1		1/400			1
1954	Gessini and Frugoni[18]	2			2/160			
1955	Murtagh *et al.*[40]	1		1	1			1
1955	Schaerer[53]	1		1	1/600			1
1955	Zaclis[71]	1		1	1			1
1955	Poblete and Asenjo[46]	2	2		2/828		1	1
1956	Brea[5]	2			2/250			
1956	Gros *et al.*[19]	1			1			
1957	Krayenbühl and Yasargil[31]	7			7			
1957	Mount and Taveras[39]	1		1	1		1	
1957	Tartarini and Nocti[65]	1			1			
1957	Wise[70]	1	1		1/397		1	
1958	Decker and Hipp[10]	1			1			
1958	Heidrich[22]	1	1		1	1		1
1958	Hook[24]	1			1			
1958	Kepes and Kernohan[28]	1	1			1	1	
1959	Rupprecht and Scherzer[49]	6			6/3,000		4	2
1959	Saltzman[50]	8	3	5	8		2	6
1959	Schiefer and Walter[55]	8	3	5	8/1,657		2	
1959	Wiedenmann and Hipp[69]	6	4	2	6/7,382		4	2
1960	Jackson and Garza-Mercado[27]	1	1		1/800			1
1960	Russell and Earle[50]	3	1	2		3/640	1	2
1961	Bingham and Hayes[3]	4		1	4/1,600		1	1
1961	Campbell and Dyken[6]	4	3	1	4/1,076		3	1
1961	Cantore and Silipo[7]	1			1			
1961	Gannon and Kaplan[10]	2	2		2		2	
1961	Lamb and Morris[32]	5	4	1	5/1,500	3	3	2
1961	Ruberti *et al.*[48]	1			1			
1961	Sanchez and Lopez-Escobar[52]	1	1		1		1	

TABLE I (*Continued*)

Year Reported	Author	No. of Cases	Sex		Diagnosis by Angiography (no. of cases /total no.)	Diagnosis at Autopsy (no. of cases /total no.)	Side	
			F	M			R	L
1961	Thomas[66]	1		1	1/200			1
1962	Baensch[2]	1			1			
1962	Lueje Casanueva[36]	1			1			
1962	Huber[25]	1			1			
1962	Madonick and Ruskin[37]	1		1	1/3,000			
1962	Passerini and DeDonato[44]	9	3	6	9/4,000		4	5
1962	Scheffner[54]	1		1	1			1
1962	Shapiro[56]	2			2			
1962	Shimo-Oku *et al.*[57]	1			1			
1962	Titrud[67]	2			2	1		
1963	Gannon[17]	1	1		1/1,200		1	
1963	Matsubara *et al.*[38]	1			1			
1963	Wollschlaeger	2	2		1/400	1/211	2	
	Summary	134	38	39	105	38	52	43

tomotic channel from the internal carotid artery proximal to the siphon to the basilar artery was noted (Fig. 1, *A* and *B*). The posterior cerebral arteries were outlined bilaterally and the right and left superior cerebellar arteries were demonstrated. The basilar artery proximal to the anastomosis could not be identified.

A few days later the patient lapsed into hepatic failure and expired. Unfortunately, the anastomosis was not recognized at autopsy, though on sectioning the brain an unusual vessel stump was noted proximal to the origin of the superior cerebellar arteries. The basilar artery between the junction of the vertebral arteries and carotid-basilar link appeared hypoplastic—a common finding associated with this anomaly.

Comment. The early shunt of contrast medium into the posterior circulation during carotid artery injection is a rather characteristic sign of the existence of a carotid-basilar anastomosis. However, since a severe degree of stenosis or an occlusion of the basilar artery may, in certain respects, re-

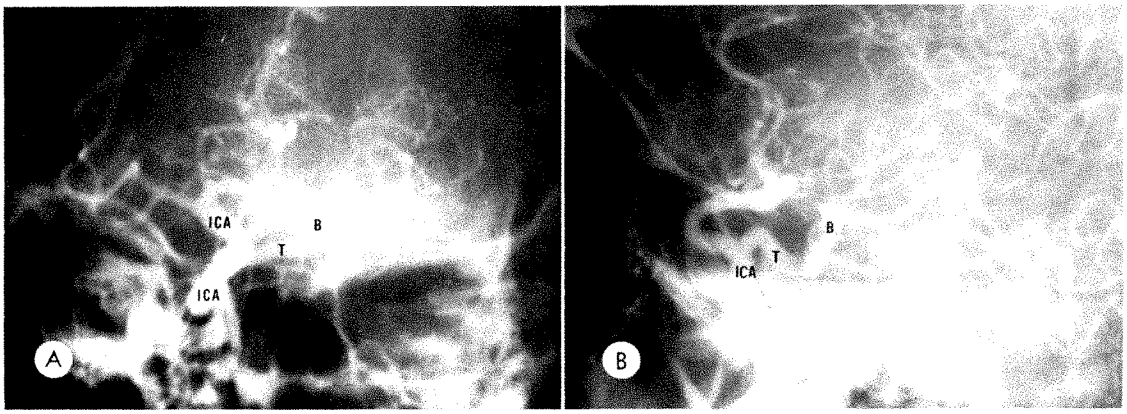

FIG. 1. (Case 1). (*A*) Anteroposterior angiogram in semi Towne projection shows the spontaneous filling of the distal basilar artery (B) and the right and left posterior cerebral arteries. The contrast filling occurred via a large anastomotic channel (T) from the right internal carotid artery (ICA). (*B*) Lateral view shows visualization of the posterior circulation. The linkage (T) from the proximal siphon of the internal carotid artery (ICA) to the basilar artery (B) is distinctly outlined.

semble the findings of this anomaly, the diagnosis of a trigeminal artery should be made only if the anastomosis between the internal carotid artery and the basilar artery is well demonstrated or if a basilar arterial occlusion is excluded by vertebral angiography.

CASE II (Hosp. # 221 518, T.G.). This 65 year old white female was admitted because of right cerebellar signs. The patient expired 1 day after admission while in a coma. At autopsy, after separation of the second and third nerves and the internal carotid artery, an unusual artery arising from the basilar artery and coursing first laterally and then anteriorly and slightly superiorly, then disappearing in the dura duplication lateral to the sella turcica, was noted. The dura was split and the vessel was traced into the cavernous sinus, where it joined the right internal carotid artery. The latter was dissected proximal to this junction, so that the total specimen was removed (Fig. 2, *A* and *B*). Postmortem angiograms (Fig. 3, *A*, *B* and *C*) showed the following variations of the normal circle of Willis: a doubling of the anterior communicating artery, an absent right posterior communicating artery and a fetal type left posterior cerebral artery, as well as thinning of the proximal basilar artery, a hypoplastic left vertebral artery and thrombosis of the right posterior inferior cerebellar artery with cerebellar infarction (Fig. 4).

Comment. Case II to our knowledge is the first in which a complete pathologic specimen revealing the anomalous vascular channel was obtained and the only case of carotid-basilar anastomosis found in our study of 211 postmortem injected brains.

DISCUSSION

In both of our cases no clinical symptoms could be connected with the presence of the anastomosis. Likewise, with very few exceptions, in most of the reported cases symptoms or pathologic changes immediately related to the entity were not observed. Heidrich,[22] in 1958, described a patient with a third nerve palsy and trigeminal

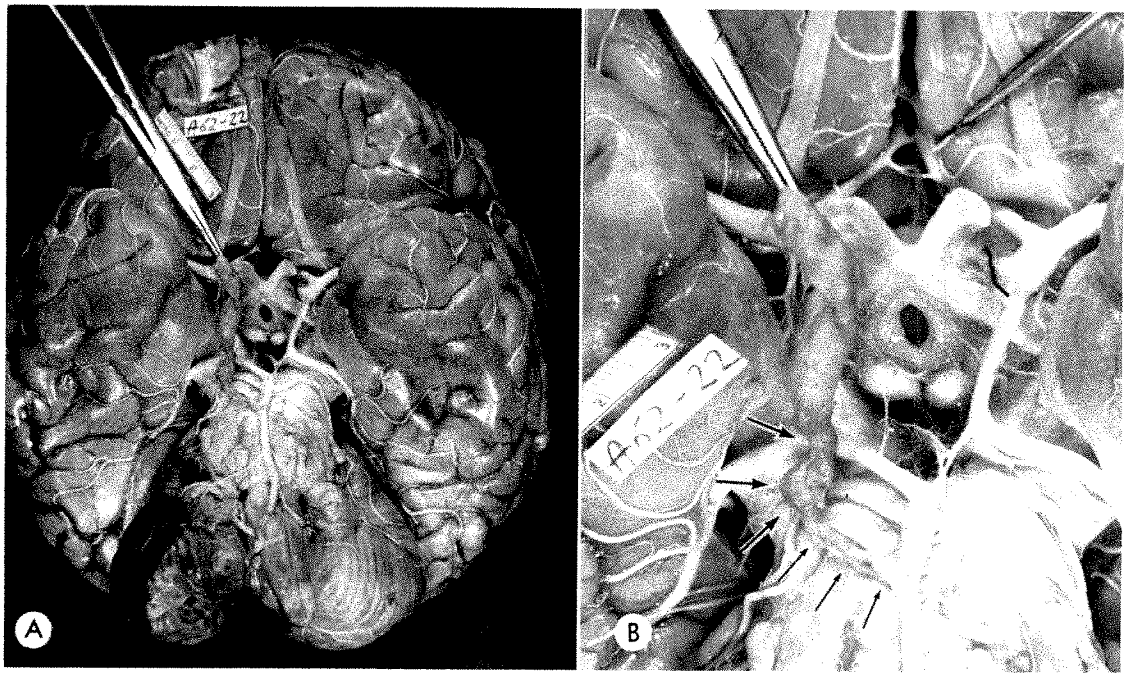

FIG. 2. Case II. Autopsy specimen. (*A*) The complete brain, with base view of the circle of Willis. The anastomotic channel has an equal lumen from the point where it pierces the dura to the junction at the basilar artery. Within the dura and in the cavernous sinus, the lumen is widened. Infarction and destruction of the right cerebellum are seen. (*B*) A close-up base view *in situ*. Arrows point to the course of the entire length of the anastomotic channel. The large arrows point to the intradural portion of the vascular anomaly. Note the increased diameter of the distal 2 cm. of the basilar artery.

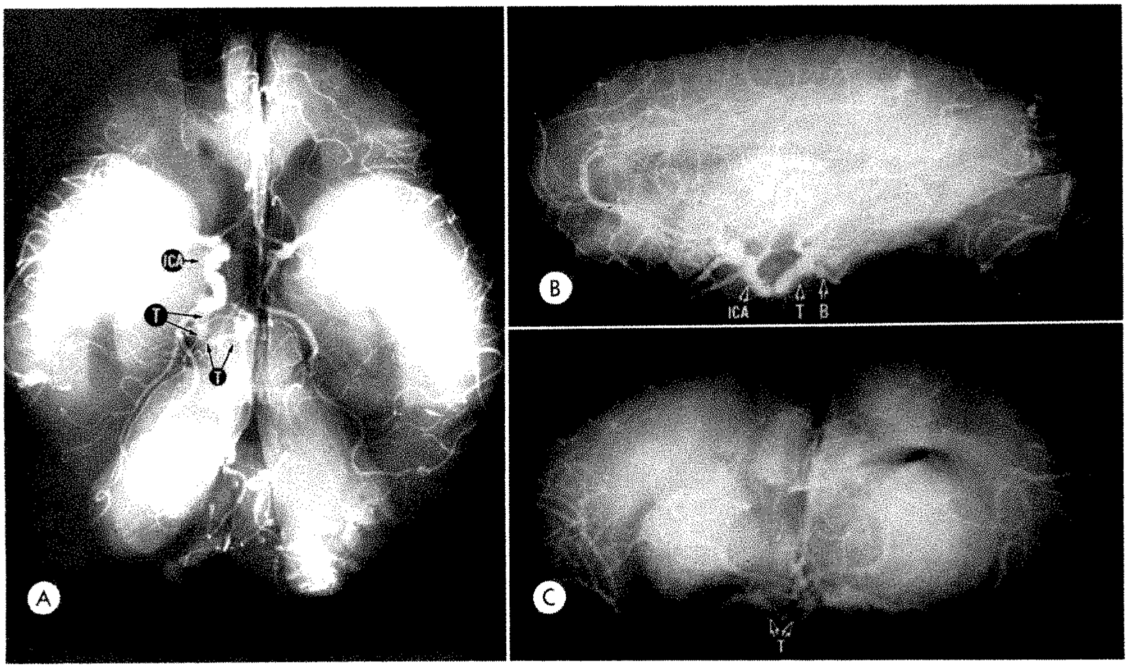

Fig. 3. (Case II). (*A*) Base view postmortem angiogram shows the anastomotic channel (T) between the right internal carotid artery (ICA) and the basilar artery, about 0.5 cm. proximal to the origin of the superior cerebellar arteries. T in the small circle points to the extradural and T in the large circle to the intradural portion of the primitive trigeminal artery. There is incomplete filling of the branches of the right posterior inferior cerebellar artery. The base view corresponds closely to the submento-occipital view, which provides a good demonstration of the junction of the carotid-basilar anastomosis and the basilar artery.[43] (*B*) Lateral view. B—basilar artery. (*C*) The anteroposterior view shows a foreshortened basilar artery. The junction of the primitive trigeminal artery (T) can be identified; the anastomosis is only faintly seen.

nerve neuralgia, in whom carotid arteriography disclosed a primitive trigeminal artery at the site of the clinical signs. Autopsy confirmed the existence of the anastomosis with otherwise negative intracerebral findings. Madonick and Ruskin,[37] in 1962, also reported a case of recurrent oculomotor paresis associated with this type of vascular anomaly.

In our series of angiographic examinations, 20 tumors and arteriovenous malformations, 9 subdural and intracerebral hematomas and 14 aneurysms were diagnosed. Mount and Taveras,[39] in 1957, reported in their paper, on intracerebral collateral circulation, a patient with an aneurysm of the internal carotid artery on the left side and a primitive trigeminal artery on the right side. After a Silverstone clamp was applied to the left common carotid artery, no complications were encountered. Passerini and DeDonato,[44] in 1962, found tumors in 6 of 9 cases with primitive trigeminal arteries and aneurysms in 2.

Kotrnetz,[30] in 1931, emphasized the importance of the knowledge of such vascular anomalies in cases of rhizotomy and hypophysectomy, where considerable difficulties may be experienced at surgery.

SUMMARY

1. A review of the literature of cases of primitive trigeminal artery reported up to early 1963 is presented.
2. Two additional cases of primitive trigeminal artery are added to the 132 already reported, and the angiographic findings are described.

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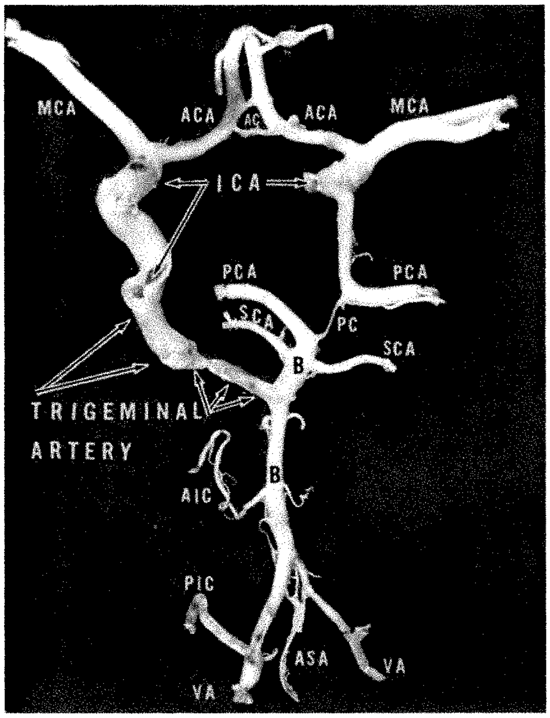

FIG. 4. Injected circle of Willis removed from the brain specimen for better demonstration of the associated anomalies. AC—doubling of anterior communicating artery; PCA—left fetal type posterior cerebral artery with origin from the left internal carotid artery (ICA); PC—hypoplastic left pars circularis posterior; MCA—middle cerebral arteries; ICA—internal carotid arteries; ACA—anterior cerebral arteries; VA—hypoplastic left vertebral artery; SCA—superior cerebellar arteries; AIC—anterior inferior cerebellar artery; PIC—posterior inferior cerebellar artery; ASA—anterior spinal artery. Upper B indicates the dilated distal basilar artery, lower B the hypoplastic proximal basilar artery. The right posterior communicating artery is absent.

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GIANT CELL (CRANIAL) ARTERITIS*

A CASE WITH ANGIOGRAPHIC ABNORMALITIES

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GIANT cell arteritis is a subacute inflammatory disease of arteries which usually occurs in adults over 55 years of age. In the earliest reported cases, the temporal arteries were most strikingly involved and the condition was labeled "temporal arteritis."[10] Later cases in which intracranial arteritis predominated became known as "cranial arteritis."[12] It was ultimately realized that these are variants of "giant cell arteritis," a condition which may be localized or relatively widespread.[6]

In the literature, the frequency of visual complications is emphasized and the involvement of intracranial arteries is documented repeatedly. Nevertheless, there is a remarkable scarcity of references to the use of cerebral angiography in studying this disease. In the present instance bilateral cerebral angiography was performed and demonstrated striking abnormalities.

Clinical diagnosis presents no serious problem if the history or physical examination points to overt evidence of inflammation of the superficial temporal arteries. In the absence thereof, signs and symptoms may be quite diverse, the clinical picture may be obscure, and the diagnosis may not be considered during life.

REPORT OF A CASE

A 56 year old white married male clerk (#31-12-78) was admitted to the University of Oregon Medical School Hospital on October 5, 1962 with symptoms of mental confusion, obtunded sensorium, and left hemiparesis. Past history revealed one or two years of gradually progressive weakness and fatigability as well as transient episodes of diplopia for 3 to 5 months and intermittent tingling dysesthesias for several weeks before onset of the current illness.

Five weeks prior to admission, the patient went to bed because of weakness and malaise. Improvement during the next 5 days was such that he returned to work for 2 days until there developed a low-grade fever and a dull aching sensation in the supraorbital and right malar regions which could be controlled by aspirin.

He was admitted to another hospital where roentgenographic examination disclosed "clouding" of the left maxillary antrum which was interpreted as evidence of sinus infection. Antibiotic therapy was instituted. At that time, neurologic examination revealed ptosis of the right upper eyelid, a mild right central facial paresis, and absent abdominal reflexes. The electroencephalogram demonstrated a left frontal slow focus. Bilateral carotid angiography was performed on October 1, 1962 and the roentgenograms were interpreted as normal. During the next 2 weeks, difficulty in reading, shuffling of the left foot and obtunded sensorium developed. Headache and intermittent fever to 102° F. continued.

Past history was negative for nervous system diseases, head trauma, or tuberculosis. The patient had been troubled intermittently for 20 years with "sinus headaches." He was referred to the Medical School Hospital for further evaluation.

Physical Examination. On admission, the patient appeared to be a thin, somewhat cachectic adult male who showed evidence of loss of recent memory, "thought blockage," and disorientation as to time and place. The temperature was 99.6° F., blood pressure 186/100, respirations 20, and pulse 72 (regular). Pupils, 3 mm. in diameter, constricted with light and convergence stimulation. Nystagmus and gross extraocular muscle weakness were absent. Visual fields were grossly intact to confrontation. There was no definite facial asymmetry, no definite speech defect and no papilledema or optic atrophy. The movements of the left extremities were weak as compared to the right. All tendon reflexes of the extremities were active symmetrically. Hoffmann's sign was nega-

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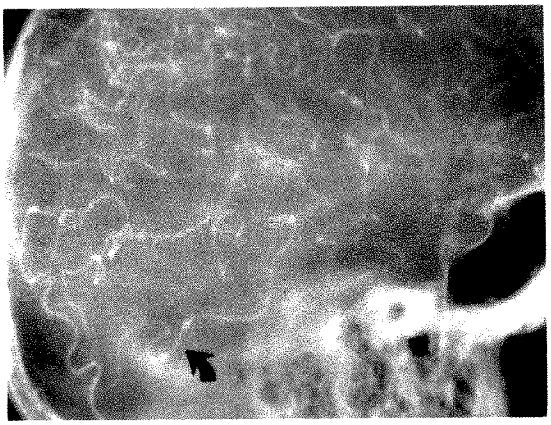

Fig. 1. Lateral carotid angiogram showing the beaded appearance of vessels in both the anterior and middle cerebral arterial arborizations. The beading is caused by stenoses and aneurysmal dilatations which occur both singly and in groups. The arrow points at an artery which is shown magnified in Figure 2.

tive. Finger-to-nose test on the left was clumsy but not ataxic. Difficulty of stereognosis in the left hand and difficulty in identifying numbers written on the skin of the palm and fingers of the left hand were noted and there was extinction to double simultaneous stimulation over the distal portions of the left extremities. Position sense was intact. Pain, touch and vibratory sensation seemed to be symmetric. The patient tended to fall to the left when standing with feet together and eyes closed. There was minimal ankle clonus on the left. Temporal, subclavian, radial, dorsalis pedis and posterior tibial artery pulsations were palpable.

Admission Laboratory Studies. The urine was yellow, slightly cloudy, with pH of 6, specific gravity of 1.014, no protein, a trace of reduction and 2+ pus. Hemoglobin was 13 gm. per cent and the white blood cell count 8,400 with 59 per cent segmented neutrophils, 2 per cent eosinophils, 2 per cent basophils, 31 per cent lymphocytes, and 4 per cent monocytes. There were 2 disintegrated cells. Spinal fluid examination demonstrated a pressure of 17 cm. of water. Analysis of fluid showed 20 white blood cells (90 per cent segmented neutrophils), 98 mg. per cent protein, 54 mg. per cent glucose and a negative VDRL.

Course. During the next 45 days, the patient remained confused or semicomatose. Fever was present almost daily, rising as high as 103.6° F. During this interval, upper eyelid ptosis and adduction weakness were noticed on the right as well as an irregular nystagmus, and loss of conjugate upward eye movement. There were loss of cremasteric reflexes, weakness of left lower face and bilateral past-pointing. Ten days after admission, right lateral rectus paresis, right lower facial paresis, and bilateral extensor plantar responses were noted. By the 22nd day after admission the muscles innervated by the right third nerve were paralyzed and 11 days later the right inferior oblique and right external rectus muscles were paretic. Two weeks after admission there occurred three generalized clonic seizures within a period of 3 days, but these did not recur after sodium dilantin therapy was instituted.

Various antibiotics were administered during hospitalization without effect. He was discharged to the care of his physician on November 20, 1962 under medication with streptomycin, isoniazid and prednisone and did not regain consciousness before he expired of bronchopneumonia 11 days later.

DIAGNOSTIC PROCEDURES

Bilateral Carotid Angiography (October 6, 1962). The upper 3 or 4 cm. of each common carotid artery was unremarkable. The posterior aspect of the left internal carotid artery immediately beyond its origin showed a 2 to 3 mm. bulge which was about 1 cm. broad at its base. A similar but less prominent bulge was noted in the same location on the right. Otherwise, the internal carotid arteries (and their ophthalmic branches) were unremarkable as were the external carotid arteries and their major divisions.

Bilaterally, the branches of the anterior and middle cerebral arteries were shown to have scattered focal areas of stenosis of varied severity intermingled with punctate radiodensities which were interpreted as contrast filled arterial ectasias (Fig. 1 and Fig. 2). The latter varied considerably in size but the largest measured about 3 or 4 mm. in maximum diameter. Some stenoses were isolated; others grouped in series to cause contour irregularity. More often stenosis was found adjacent to ectasia. Occasionally, stenoses and ectasias alternated, giving the appearance of a somewhat irregular string of beads. The lesions were most apparent in the late arterial phase and seemed slightly more prominent on the right. The venous phase was unremarkable.

Since the lesions were so widely distributed in the carotid system, the possibility that vessels elsewhere in the body might be involved was considered. The pathologic nature of the condition was not suggested.

The outside angiograms of October 1, 1962 were re-examined. In retrospect, it was evident that there was no interval change. The ability to recognize the lesions in the more recent study was apparently due to a difference in technical quality of the two examinations.

Ventriculography (October 12, 1962). Mild generalized enlargement of the third and lateral ventricles.

Biopsy of Parietal Cortex via Burr Hole (October 12, 1962). This was done in conjunction with the ventriculography with negative results.

Gastrocnemius Biopsy (October 17, 1962). Normal tissue.

LABORATORY FINDINGS

Extensive laboratory evaluation during hospitalization showed the following abnormal findings: two plus protein, 4 plus white blood cells, and 2 plus red blood cells on some of the urinalyses. The white blood cell counts ranged up to 19,553 with normal differentials and some of the sedimentation rates were as high as 32 mm. Bentonite flocculation tests were positive twice. Five spinal fluid examinations showed a range of 17 to 144 white blood cells with 9 to 100 per cent lymphocytes and 0 to 91 per cent neutrophils. Spinal fluid protein varied from 51 to 100 mg. per cent.

Normal laboratory determinations during hospitalization included hematocrit, hemoglobin, red blood cell count, LE preparations, urine porphyrins, fasting blood sugar, serum electrolytes, blood urea nitrogen, serum protein, alkaline phosphatase, spinal fluid sugar and protein, and serum electrophoresis. In addition, acute and chronic phase serums failed to demonstrate an increased titer when tested for 12 pathogenic viruses.

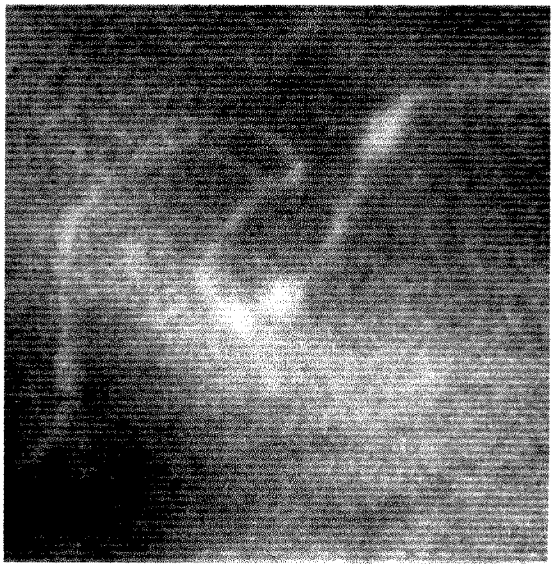

Fig. 2. Magnified photograph of artery indicated by arrow in Figure 1. The gross contour irregularity seen here is typical of the abnormality found in many of the leptomeningeal vessels.

PATHOLOGY FINDINGS

Autopsy examination was performed in another city and formalin fixed specimens of gross tissue were delivered to us for additional study. These consisted of the brain, heart, one adrenal gland, two wedges of lung, one pole of each kidney, one piece each of liver and spleen.

Gross. The brain weighed 1,590 gm. There was slight meningeal thickening over the base of the brain. The meninges were free of purulent collections. The external contours of the cerebral hemispheres, cerebellum, and brainstem were unaltered. Minimal arteriosclerotic plaque formation was present in both the right and left internal carotid arteries. The leptomeningeal vessels coursing over the cerebral hemispheres demonstrated numerous slight focal aneurysmal dilatations (Fig. 3). No occlusions or marked stenoses were identified grossly in any of the intracerebral vessels. Serial section of the cerebral hemispheres showed symmetric mildly dilated ventri-

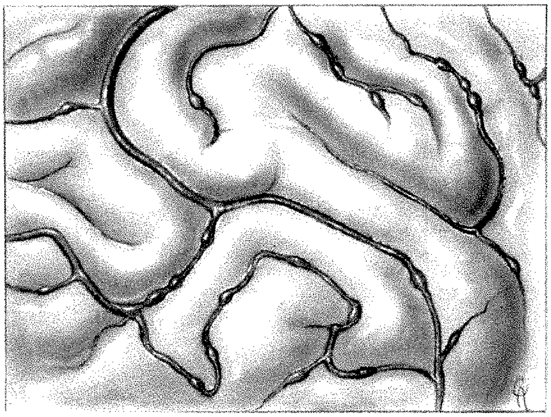

FIG. 3. Composite drawing of typical lesions seen on the surface of the cerebrum at autopsy. Stenoses and dilatations were found singly or grouped in series.

cles. Multiple focal areas in which the outer cortical ribbon was indistinct and poorly defined were identified in the left parietal lobe, left frontal lobe, and right and left temporal lobes. There was associated marked softening of both the gray and white matter in these regions. The areas of infarction measured from 4 to 16 mm. in diameter. In addition, multiple, irregular-shaped, focal cystic infarcts were present in the pons, right and left brachia conjunctiva, left caudate nucleus, and in the left globus pallidus. These cystic infarcts measured from 2 to 7 mm. in diameter.

The heart weighed 330 gm. The left ventricle measured up to 17 mm. in thickness. There was rather marked fibrosis throughout the anterior portion of the interventricular septum and in the anterior wall of the left ventricle. An old occlusion was identified in the anterior descending branch of the left coronary artery at a point 1 cm. distal to its origin. Manual compression of the wedges of lung tissue elicited a moderate amount of slightly cloudy fluid. The remainder of the tissue examined was grossly unremarkable.

Microscopic. Sections were taken from the peripheral aspects of the parietal lobes for microscopic examination of the leptomeningeal vessels. Gross sections taken through these vessels frequently showed greatly thickened arterial walls with consequent marked diminution of lumen size (Fig. 4*A*). The thickening was due to granulomatous proliferation replacing the intimal and medial layers and obliterating the internal elastic membrane. The proliferative element was composed of connective tissue fibers admixed with fibroblasts, lymphocytes, histiocytes and occasional multinucleated giant cells (Fig. 4*B*). No necrosis was identified.

Longitudinal sectioning of the leptomeningeal arteries sometimes showed areas in which the proliferative process was confined to the intimal layer, while examination of other areas disclosed a greater or lesser degree of medial involvement. Often both variants could be seen in the same section (Fig. 4, *C* and *D*). It is suggested that these areas of granulomatous proliferation and mural destruction may represent focal regions of weakness which, in the presence of physiologic intraluminal hydrostatic pressure, distend to form small aneurysmal dilatations.

Sections were taken through all of the brain infarctions mentioned above. They showed a focal loss of parenchyma often associated with numerous macrophages as well as some increase in the number of astrocytes at the margins of the infarcts.

Study of the extracerebral tissues confirmed the old myocardial infarction and demonstrated a marked bronchopneumonia. Sections of liver, spleen, kidney, and adrenal gland were essentially unremarkable histologically. None of the arteries in these organs showed the pathologic changes described in the leptomeningeal arteries.

DISCUSSION

The histologic changes are those of giant cell arteritis, a condition which is well known to involve vessels in widely varying regions of the body.[7] The relative paucity of giant cells in the lesions in this case does not suggest a differential diagnosis.[9] The occurrence of intracranial aneurysms in

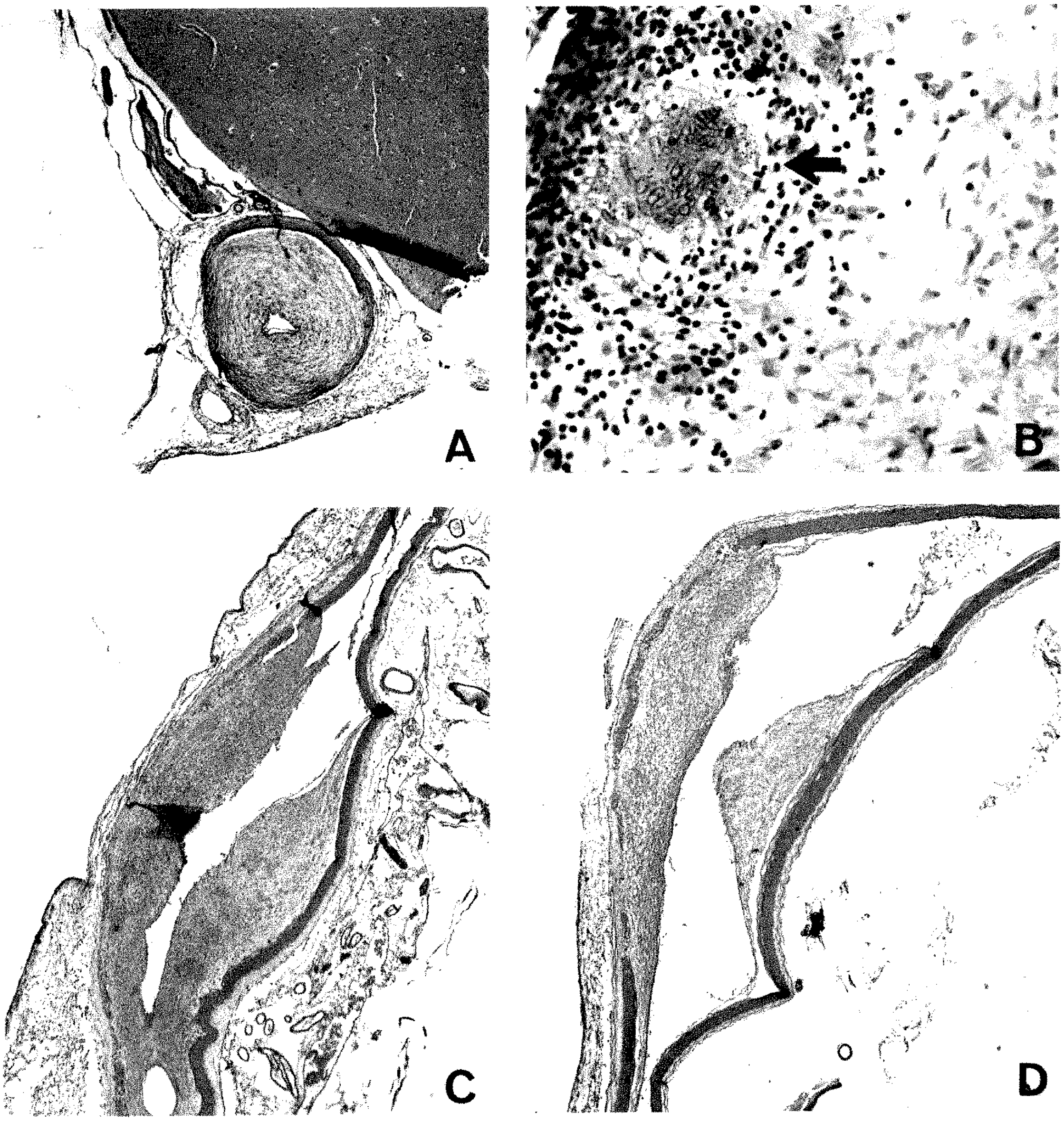

Fig. 4. (*A*) Cross section of leptomeningeal artery. Note marked thickening of intima and partial replacement of media by granulomatous proliferation (×24). (*B*) Higher magnification in area of granulomatous proliferation showing a prominent multinucleated giant cell (arrow) surrounded by a lymphocytic infiltration (×150). (*C* and *D*) Longitudinal sections through areas of aneurysmal dilatation. In some areas the granulomatous tissue is confined to the intima. In others it partly or completely replaces the media (×24).

some of these cases has been described previously;[1,6] however, from the descriptions given, these appeared to be unrelated to the giant cell arteritis and are in no way similar to the proliferative aneurysmal dilatations described in this report which more closely resemble the "false aneurysms" described by Cohen.[3] Many theories as to etiology have been advanced.[7] Our case contributed nothing in this regard.

In the literature references to cerebral angiography of patients with giant cell arteritis are rare. One report of a bilateral examination briefly noted "diffuse arteritic changes" on the right side.[18] Another mentioned stretching of the carotid siphon, narrowing of the anterior cerebral artery, and tortuosity along with narrowing of branches of the sylvian group.[8] In a monograph on temporal arteritis, it was stated that "cerebral angiograms have sometimes revealed irregularities of caliber, distinctive narrowness or striking tortuosity of certain arteries."[16]

One report was found in which a detailed angiographic description was given as follows: Abnormalities on the right side included an area of stenosis below the siphon and irregularities of the carotid artery in the siphon, the smallest of which suggested mural deposits. The ophthalmic artery was abnormally small. Anterior and middle cerebral arteries were unremarkable. The left internal carotid artery and its branches were quite similar to those on the right. As to the external carotid arteries, the right one was generally irregular in caliber as were some of its major branches. The left external carotid artery *per se* was well demonstrated but some of its major branches were poorly visualized if at all. A pneumoencephalogram of the same patient disclosed ventricles of average size, slightly widened and somewhat rounded. There was also a spotty distribution of coarsened subarachnoid spaces.[5]

To the authors' knowledge, the angiographic findings reported in this paper are unprecedented in the literature. Although it was possible to establish the presence of a bizarre widespread cerebral arterial abnormality and although the possibility that the condition might be found elsewhere in the body was raised, a specific diagnosis could not be offered.

Classic early clinical descriptions focused attention on involvement of the temporal arteries which were painful, palpably indurated and tortuous.[10,11] More recent reports of less classic clinical presentations of this disease have demonstrated its protean nature and emphasized the difficulty of diagnosing its diverse forms. Such reports are relatively infrequent.[2,4,14,17,18] At least one author has attempted to categorize the occult forms of giant cell arteritis into specific syndromes.[15] The variability of clinical manifestations is easily understood in view of the varied distribution of arterial lesions. In the present instance the clinical picture was sufficiently atypical to be misleading and the precise diagnosis was not made until the postmortem brain sections had been studied.

SUMMARY

The authors have presented a clinically atypical case of giant cell arteritis in which the lesions were confined largely to the leptomeningeal arteries.

Diagnostic studies included bilateral cerebral angiography by which striking abnormalities were discovered. These are described and illustrated and are believed to be without precedent in the literature.

Gross and microscopic features of leptomeningeal arteries obtained at postmortem examination are also described and illustrated. An attempt is made to correlate the pathologic and angiographic abnormalities.

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CHANGES IN THE ANTERIOR CHOROIDAL ARTERY IN INTRACRANIAL LESIONS*

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THE anterior choroidal artery provides diagnostic clues which are often useful and sometimes crucial, and yet this vessel has received relatively little attention in the extensive literature now available on cerebral angiography. Its location under the base of the brain near the tentorial notch exposes it to pressure from mass lesions both above and below the tentorium. Its central location in the chiasmatic and interpeduncular cisterns exposes it to pressure from mass lesions in the frontal fossa as well as those in the occipital and temporal regions. Our purpose is to describe changes in the appearance of the anterior choroidal artery which are of unique help in localizing intracranial lesions.

ANATOMY

In lower animals the choroid plexus of the lateral ventricle is supplied mainly by branches from the posterior cerebral artery. As the cerebral hemisphere enlarges, the lateral ventricle enlarges and develops an inferior horn, and the choroid plexus elongates and extends into the inferior horn. The inferior portion of the plexus receives its blood supply from an anterior choroidal vessel, or series of smaller vessels, arising from the anterior division of the internal carotid artery.[1] In the lower animals, the anterior choroidal artery anastomoses freely with branches from the posterior cerebral artery over the base of the brain, but in man these anastomotic branches are small and the larger branches of the artery are those supplying the choroid plexus.

The posterior choroidal arteries, arising from the posterior cerebral artery, are smaller and less readily identified roentgenographically than the anterior choroidal artery. They supply mainly the choroid plexus of the third ventricle and lateral ventricles, and their anatomy has been well described by Duret[3] (whose name was applied to them at one time) and, in a more recent article, which includes the roentgenographic study of the vessels, by Galloway and Greitz.[5] There are two main divisions of the posterior choroidal arteries: the posterior internal, or medial, choroidal artery, which arises from the proximal portion of the posterior cerebral artery and passes forward around the brain stem and along the roof of the third ventricle to supply the choroid plexus of that ventricle; and the posterior external, or lateral, branches (usually at least two are present), which supply the choroid plexus of the body of the lateral ventricle and part of the plexus of the inferior horn. These vessels are more frequently identified when the anterior choroidal artery is small (Fig. 1 and 2).

The roentgen anatomy of the anterior choroidal artery in health and disease has been described by Sjögren,[10] Ecker and Riemenschneider,[4] and in less detail by Morello and Cooper,[8] and by Lenzi.[7] The origin of the artery varies, but it arises most often from the internal carotid artery just above the origin of the posterior communicating artery. In anteroposterior projection (Fig. 3), it is visualized arising from the medial side of the internal carotid artery. At first it curves gently, convex medially and upward, in the chiasmatic and interpeduncular cisterns, around the uncus, then turns laterally and kinks as it enters the inferior horn, then curves, convex laterally, and courses posteriorly. Its appearance varies according to the beam direction, being least foreshortened in the half-axial projection. In the lateral projection (Fig. 4), the plane of the anterior choroidal artery is seen to parallel that of the larger vessels in the sylvian fissure. Its proximal, cisternal portion, that curving over the uncus, usually takes a gently curving or, more often, gently undulating course posteriorly, and then turns sharply as it enters the inferior horn of the lateral ventricle. It can usually be followed into its ventricular (plexal) portion, but the network of the choroid plexus is not commonly visualized roentgenographically. In most cases the anterior choroidal artery is easily differentiated from the posterior cerebral artery because it is much smaller; it is about the same size as the smaller branches of the middle cerebral artery.

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Stereoscopic studies are extremely useful in identifying the vessel and in differentiating it from the smaller branches of the middle cerebral artery. Of 100 consecutive carotid artery examinations, we were able to identify the anterior choroidal artery in the lateral study in 99, but stereoscopic views were necessary in 26 for definite identification. In the one case in which the artery was not seen, a large lateral posterior choroidal branch was present, coursing to the region of the glomus. When stereoscopic arteriograms were necessary, they were taken in the late arterial phase, and the

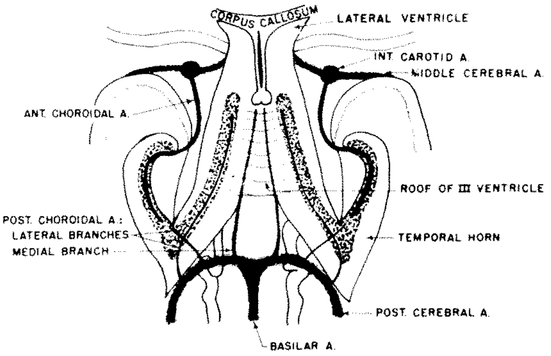

Fig. 1. Diagram of the blood supply of the choroid plexus of the lateral ventricle. The ventricles are exposed from above.

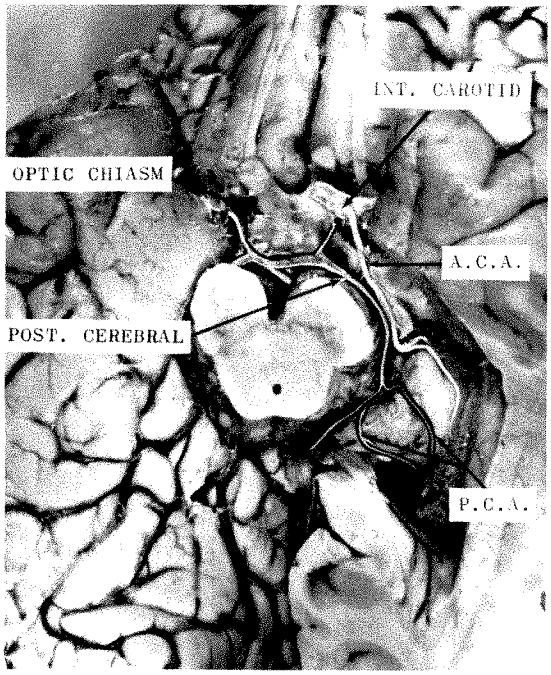

Fig. 2. The brain from below. Part of one temporal lobe has been removed to expose the choroid plexus in the inferior horn of the lateral ventricle. Two posterolateral choroidal arteries (P.C.A.) enter the plexus in the region of the glomus. A.C.A.—anterior choroidal artery.

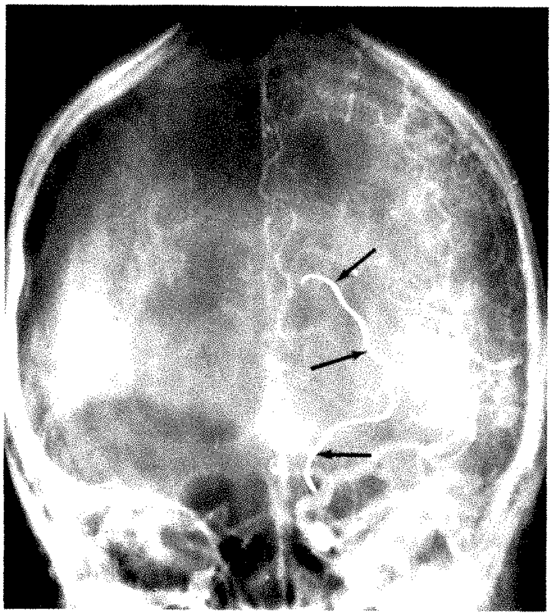

Fig. 3. Anteroposterior roentgenogram of a normal anterior choroidal artery which can be followed into the body of the lateral ventricle.

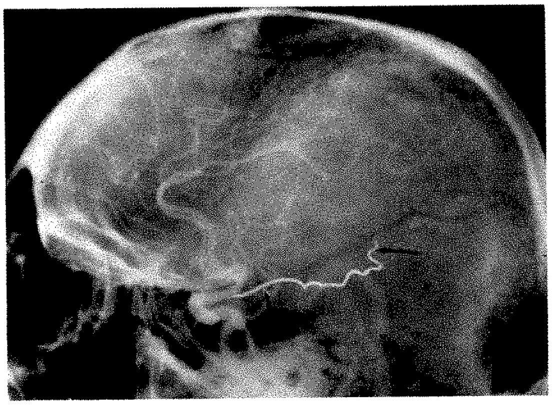

FIG. 4. Normal position of the anterior choroidal artery in the lateral view. The posterior temporal and parietal branches of the middle cerebral artery are absent.

anterior choroidal artery was partially obscured by branches of the middle cerebral artery. In the anteroposterior roentgenograms, the anterior choroidal artery was not identified in 12 of the 100 examinations. Identification was made difficult by lack of contrast or by the patient's chin being elevated too much with resultant foreshortening of the artery. The vessel is usually visualized earlier than other branch arteries and may be filled when only 1 to 2 ml. of contrast medium has been injected. Sjögren[10] has always found it possible to identify the artery when using rapid serial roentgenography.

According to Lenzi,[7] the maximum normal length of the anterior choroidal artery is 3 cm., and when the artery is longer than this some abnormality is present, usually a tumor—most often an intraventricular meningioma or deep-seated glioma. Certainly the anterior choroidal artery may be unduly long if it is feeding a tumor or is stretched by a tumor; but in many, indeed in a majority, of our normal cases, the artery measured more than 3 cm. on the roentgenogram while the other findings and the clinical course ruled out a tumor. In most of these cases, the artery appeared normal except for its unusual length, and we believe that the proportion of the artery visualized angiographically varies according to the degree of filling, and hence depends mainly on the timing of the exposures after injection.

THE ABNORMAL ANTERIOR CHOROIDAL ARTERY

Masses in certain intracranial sites affect the course of the anterior choroidal artery in characteristic ways. The artery may be affected either because it is feeding a tumor, or because a tumor lies close to it and stretches it, or because the origin of the anterior choroidal artery from the internal carotid artery is distorted due to herniation through the tentorial notch. The changes in

TABLE I

CHANGES IN THE ANTERIOR CHOROIDAL ARTERY CAUSED BY INTRACRANIAL MASS LESIONS

	Cisternal Portion	Ventricular Portion
Tentorial herniation		
Ascending herniation of brain stem and/or cerebellum due to masses in the posterior fossa	Upward displacement	
Descending uncal herniation	Straightened and displaced medially over the tentorial notch, or downward bulge	
Frontal fossa, parasagittal, or midbrain lesions	Flattened and depressed	
Occipital masses	Compressed from posteriorly in concertina manner	
Temporal lobe tumor	Stretched and displaced medially	Stretched
Temporal lobe edema	Medial displacement; not stretched	Not Stretched
Obstruction of temporal horn	Stretched	Not Stretched

the anterior choroidal artery which may be caused by intracranial mass lesions are listed in Table I.

Masses in the posterior cranial fossa with upward herniation of the brain stem or cerebellum, or both, may cause lateral displacement of the cisternal portion of the anterior choroidal artery and/or may cause the artery to bulge sharply upwards. Most of the course of the visualized anterior choroidal artery in these cases is taken up by a single arc, convex upwards, in contrast with the usual undulating course of the vessel. The reason for this abnormal course is probably upward displacement of the uncus by a portion of the superior aspect of the cerebellum herniated through the tentorial notch. Figure 5 is an arteriogram of a 34 year old man with a bronchogenic carcinoma. He was admitted because of severe bifrontal headaches for a period of 10 days. The lateral arteriogram suggested a posterior fossa lesion and this was confirmed several days after the arterial study, when the patient died and a metastatic cerebellar tumor was found, with upward herniation of the superior portion of the cerebellum.

Downward herniation of the anterior choroidal artery through the tentorial notch may occur when the uncus of the temporal lobe is forced over the edge of the tentorium in patients with supratentorial tumors or hemorrhages, or even with generalized increased intracranial pressure. Usually, the cisternal portion of the artery is straightened and displaced medially over the tentorial notch, but it may even arch downward (Fig. 6).

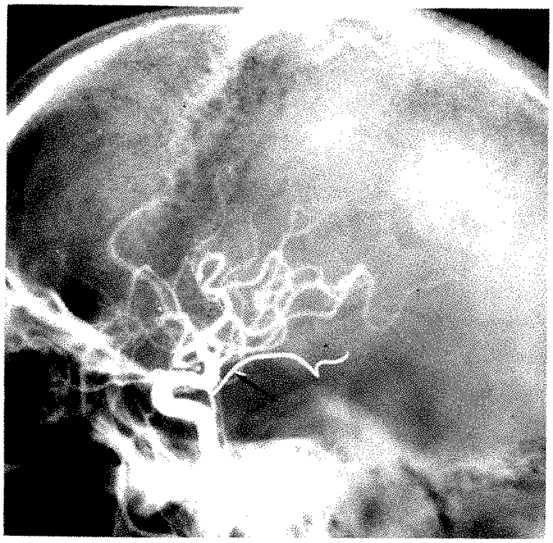

Fig. 5. Upward displacement of the anterior choroidal artery caused by a cerebellar tumor.

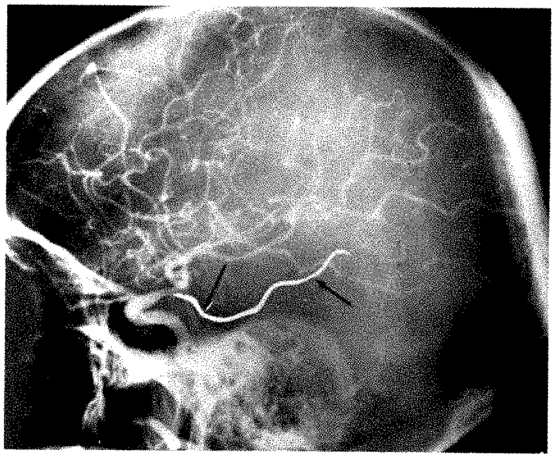

Fig. 6. Downward herniation of the anterior choroidal artery through the tentorial notch in a patient with a subdural hematoma.

Frontal, parasagittal, and midbrain masses tend to depress the anterior choroidal artery so that it runs directly posteriorly with a flattened appearance, or even (if there is herniation through the tentorial notch) courses in a shallow arc that is convex inferiorly. In a 45 year old man suspected of having a posterior fossa lesion, arteriograms (Fig. 7, *A* and *B*) showed evidence of hydrocephalus, but the flattening of the anterior choroidal artery pointed to a supratentorial lesion, and on ventriculography a mass was shown to lie mainly above and posterior to the third ventricle.

Occipital masses (*i.e.*, those in the occipital lobe and the adjacent parts of the parietal and temporal lobes) produce a characteristic deformity, in which the anterior choroidal artery is compressed from posteriorly in concertina fashion (Fig. 8, *A* and *B*). The cisternal portions of the artery take a zigzag up-and-down course in most cases; in others the artery is pushed forward and upward so that its general direction is su-

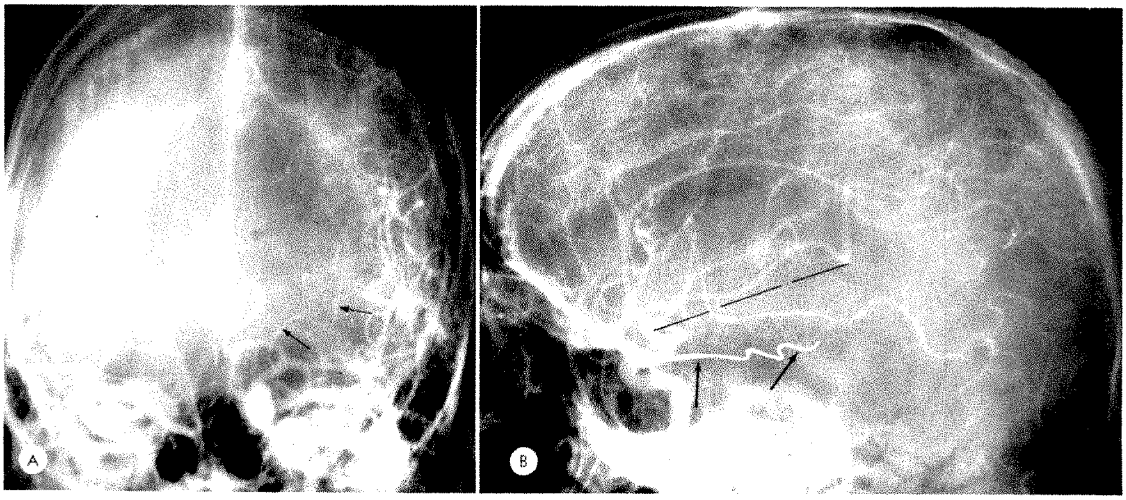

Fig. 7. (*A* and *B*) Arteriograms of a patient with a midline tumor above and posterior to the third ventricle. Hydrocephalus was secondary to downward pressure on the aqueduct. The straightened and elevated anterior cerebral artery and outward displacement of the middle cerebral artery indicate hydrocephalus. The anterior choroidal artery, rather than lying parallel with the artery of the angular gyrus (which identifies the base line of the sylvian fissure), is flattened and depressed, indicating pressure from above.

perior or posterosuperior rather than posterior.

Temporal lobe masses (except those lying far posteriorly, which behave as occipital masses), characteristically cause stretching of the anterior choroidal artery, with loss of its usual gentle undulations. The stretching is so characteristic of temporal lobe tumors that if the other roentgenographic signs suggest a temporal lobe tumor but the anterior choroidal artery is not stretched, some other diagnosis must be considered—such as a tumor outside the temporal lobe (a sphenoid-ridge or middle-fossa meningioma), or an obstructed temporal horn, or edema of the temporal lobe. The appearance of the anterior choroidal artery may be the only diagnostic clue in early tumors of the temporal lobe, when there is as yet no displacement of the larger vessels. In a patient with temporal lobe seizures of recent onset, the cisternal portion of the anterior choroidal artery was displaced medially in the anteroposterior arteriogram and slightly stretched in the lateral study (Fig. 9, *A* and *B*). A year later (Fig. 10), the tumor was more obvious. In the lateral view, the plexal portion of the vessel was stretched and there was positive displacement of the middle cerebral vessels.

Edema of the temporal lobe secondary to hemorrhage or infection can simulate a malignant tumor on arteriography, and the appearance of the anterior choroidal artery may be crucial in making the diagnosis. A 53 year old diabetic man was admitted because of dysphasia and progressive right hemiparesis of 1 week's duration. His temperature ranged between 101°F. and 103°F. On carotid arteriograms (Fig. 11, *A* and *B*), the middle cerebral group was elevated and displaced slightly medially; but the anterior choroidal artery, although medially displaced, was not stretched, and in the lateral view showed prominent undulations suggesting a posterior mass. At operation, an abscess was found deep in the posterior part of the temporal lobe. Arteriograms 3 weeks later (Fig. 12, *A* and *B*), after drainage of the abscess, showed the arteries in essentially normal position. When the whole temporal lobe is enlarged, the cisternal portion of the anterior choroidal artery may be medially displaced and even overlie the tentorial notch; but the portion of the vessel lying within the inferior horn is subjected to equal pressure from all sides and will not be stretched.

Edema of the temporal lobe secondary to vascular lesions causes similar displace-

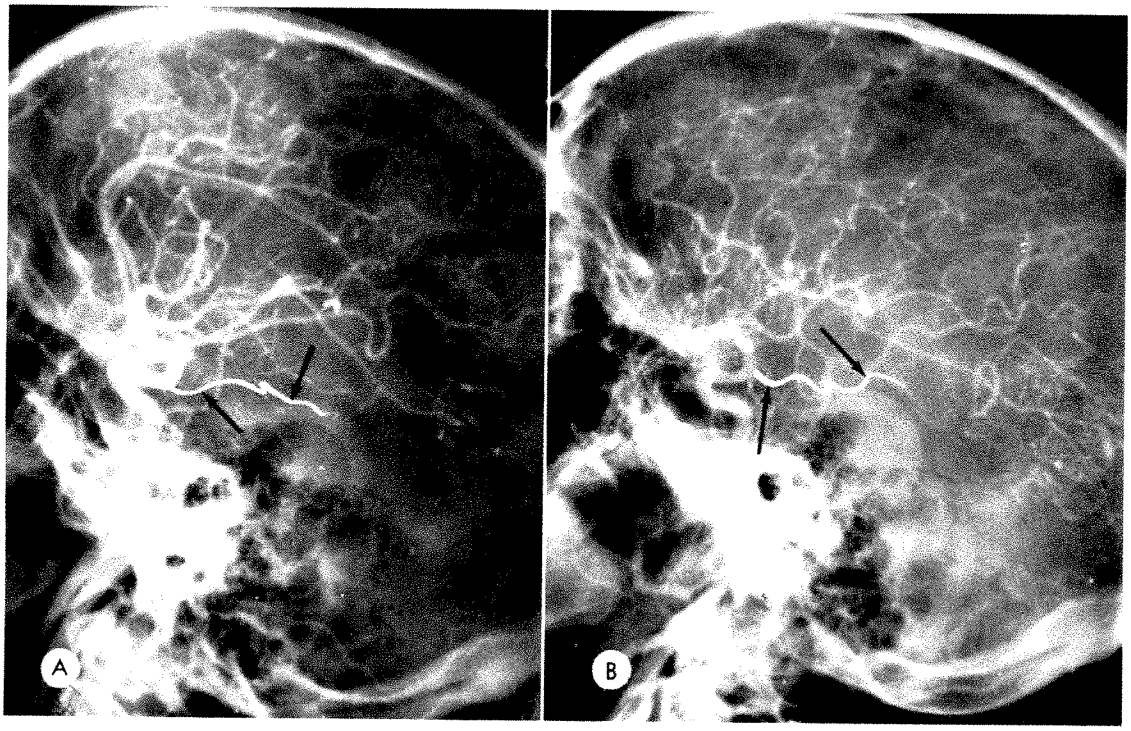

Fig. 8. (*A*) Left carotid arteriogram showing a normal anterior choroidal artery. (*B*) A right carotid arteriogram in the same patient shows the anterior choroidal artery to be displaced in a concertina manner by an occipital lobe vascular tumor.

ment of the middle cerebral and anterior choroidal arteries without stretching the anterior choroidal artery, and, even when a hematoma is present within the temporal lobe, this artery is rarely stretched. Malignant tumors in the tip and central portions of the temporal lobe almost invariably stretch the anterior choroidal artery if they are large enough to cause any arteriographic abnormality at all, even when the larger arteries are unaffected.

Enlargement of the inferior horn of the lateral ventricle due to either complete or incomplete obstruction, secondary to adhesions[2,9] or pressure from intra- or extraventricular tumors,[6] may be mistaken for edema or a tumor of the temporal lobe on arteriograms because the middle cerebral branches are elevated and the anterior choroidal artery is displaced medially. But, whereas in tumors of the temporal lobe the whole of the anterior choroidal artery is usually stretched and in edema this artery is rarely stretched, in obstructions of the inferior horn, the cisternal portion of the anterior choroidal artery is stretched over the displaced uncus but the plexal portion (which lies within the dilated horn) is not stretched at all. Figure 13, *A* and *B* shows these findings in a patient with an obstructing meningioma of the body of the lateral ventricle which resulted in unilateral hydrocephalus of the inferior horn. Roentgenograms in the venous phase showed no tumor blush, and the meningioma was no doubt fed mainly from posterior choroidal vessels.

Vascular occlusion, with secondary anastomotic channels, is suggested by obvious branching of the anterior choroidal artery on arteriograms. A series of branches normally arises along the length of the anterior choroidal artery, going to the uncus, choroid plexus, basal ganglia, and midbrain; but these are small and rarely well demonstrated roentgenographically. A 37 year old man was admitted to the hospital with a short history of intermittent involuntary movements of the left lower limb and mild left hemiparesis. Because of large temporal branches coming from the anterior cho-

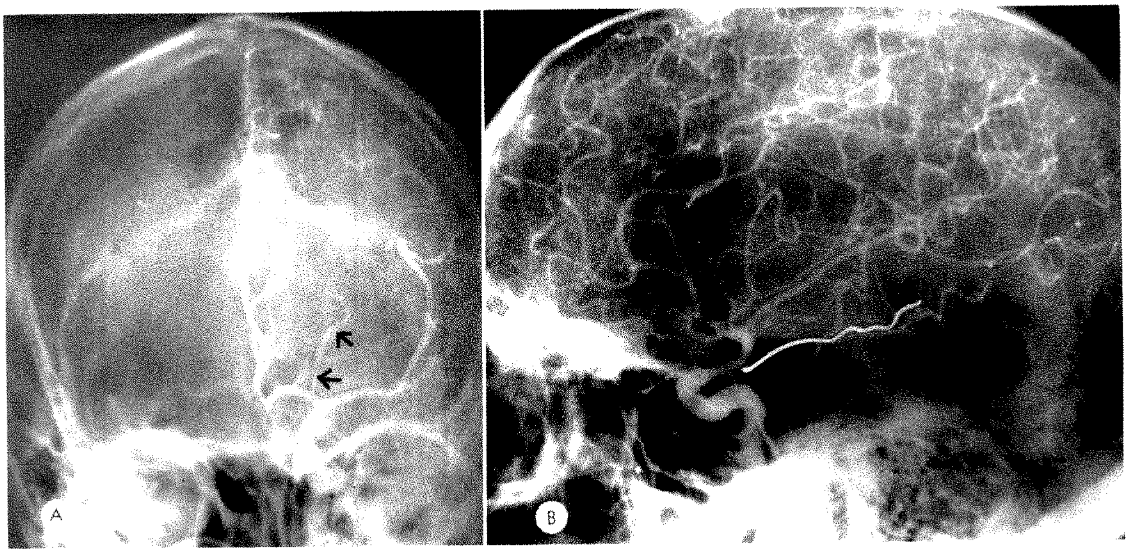

Fig. 9. (*A* and *B*) Slight stretching and medial displacement of the anterior choroidal artery in a patient with a tumor beginning in the tip of the temporal lobe.

roidal artery (Fig. 14, *A* and *B*), the possibility of basilar-artery disease was suggested by the radiologist. This was confirmed by vertebral arteriography, and the patient has since developed carotid artery occlusion. Similar changes have been seen in 2 other patients.

SUMMARY

The normal anatomy of the anterior choroidal artery is described, along with the roentgenographic appearance of the artery in normal and abnormal states. The changes caused by intracranial mass lesions are listed in Table 1. The proximal portion of the artery, coursing over the uncus of the temporal lobe, lies at the base of the brain in the chiasmatic and interpeduncular cisterns and is easily displaced and stretched by mass lesions in/or displacing the uncus. The distal portion of the artery lies in the inferior horn of the lateral ventricle and is displaced with ventricular displacement but is stretched only with mass lesions, usually malignant tumors, within the temporal lobe.

Prominent branching of the artery suggests cerebral vascular disease.

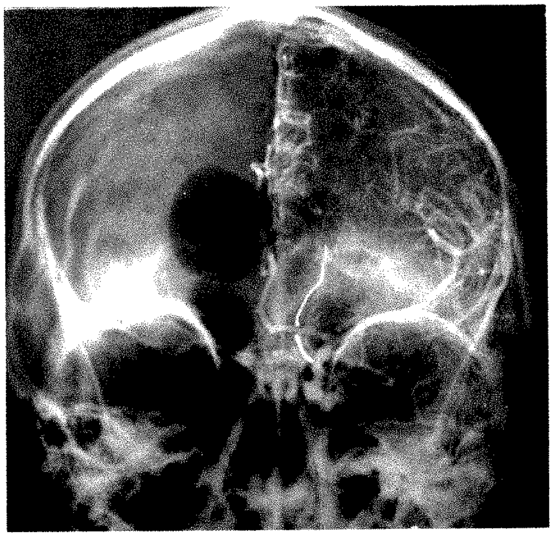

Fig. 10. Anteroposterior arteriogram 1 year later of the same patient shown in Figure 9, *A* and *B*, demonstrating extensive growth of the temporal lobe tumor.

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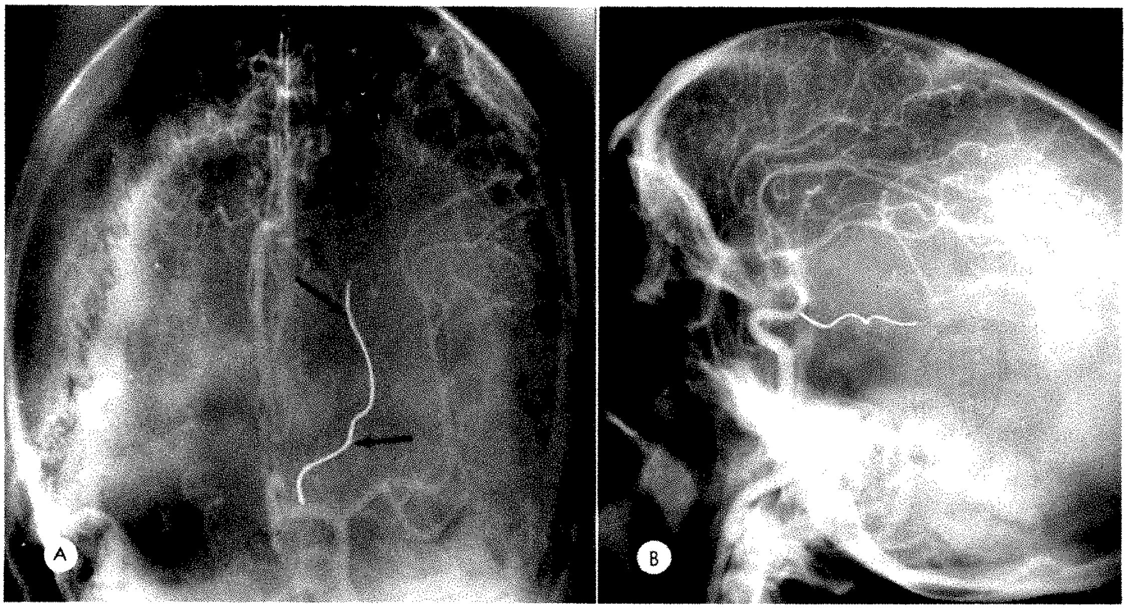

Fig. 11. (*A* and *B*) Arteriograms of a patient with an abscess deep in the posterior temporal region (shaded oval region). Edema of the temporal lobe caused upward displacement of the middle cerebral vessels but no stretching of the anterior choroidal artery.

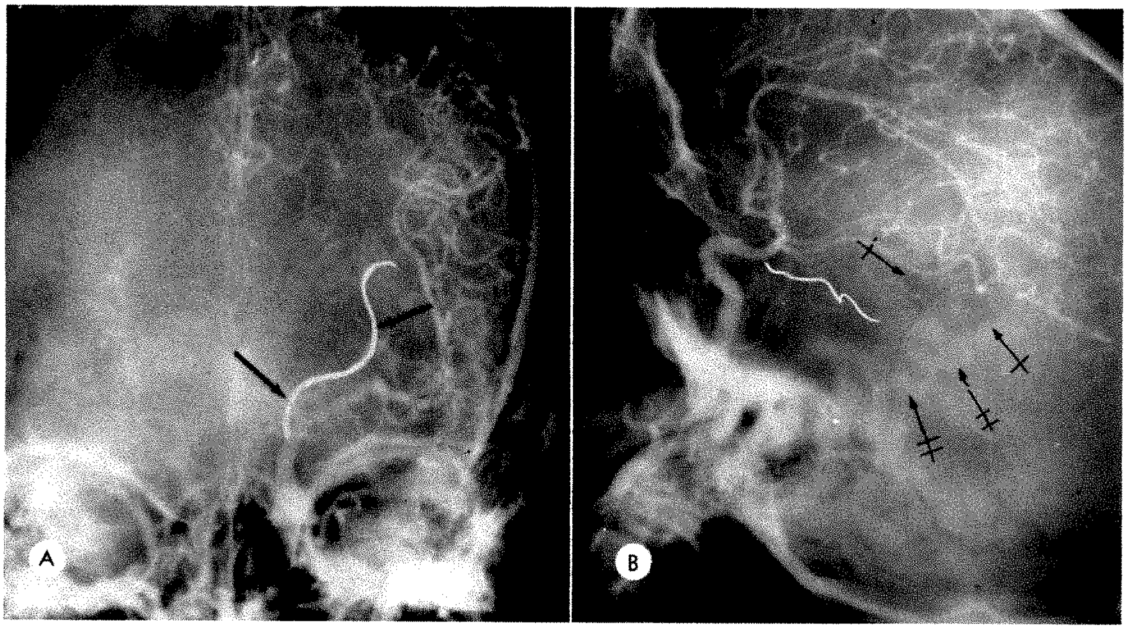

Fig. 12. (*A* and *B*) Arteriograms made 3 weeks after drainage of the abscess. ←+ marks the edge of the operative defect. ←‡ indicates a small amount of thorotrast placed in the abscess cavity at surgery.

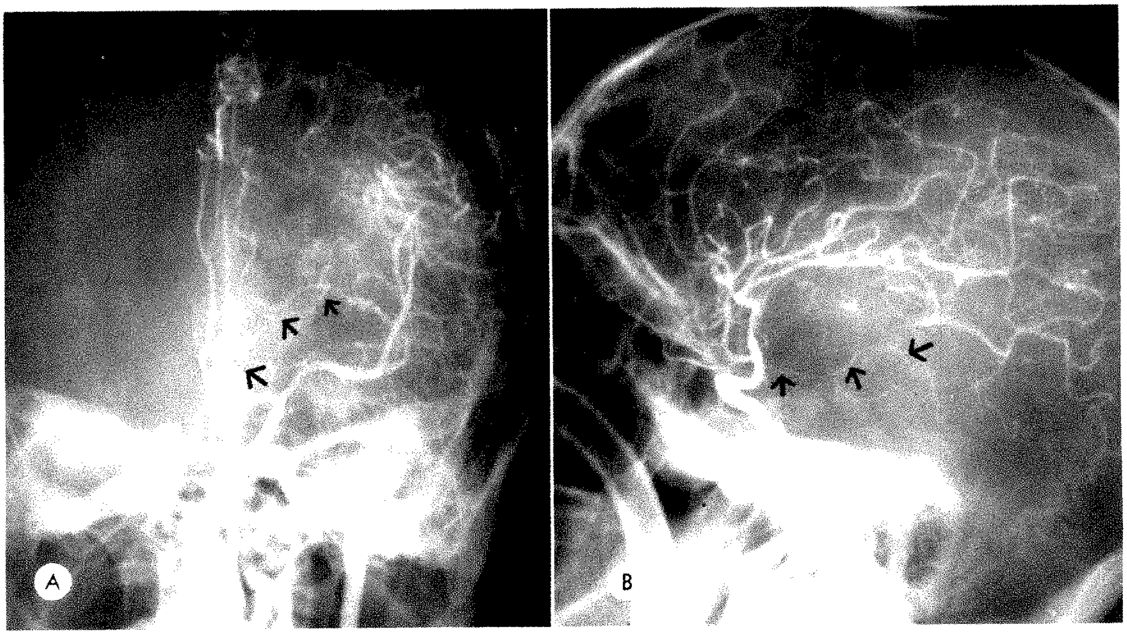

Fig. 13. (*A* and *B*) Localized obstructive hydrocephalus of the inferior horn of the lateral ventricle secondary to a meningioma in the body has caused medial displacement and slight stretching of the cisternal portion of the anterior choroidal artery, but the plexal portion of the artery appears normal. (By courtesy of *Acta radiologica*.[6])

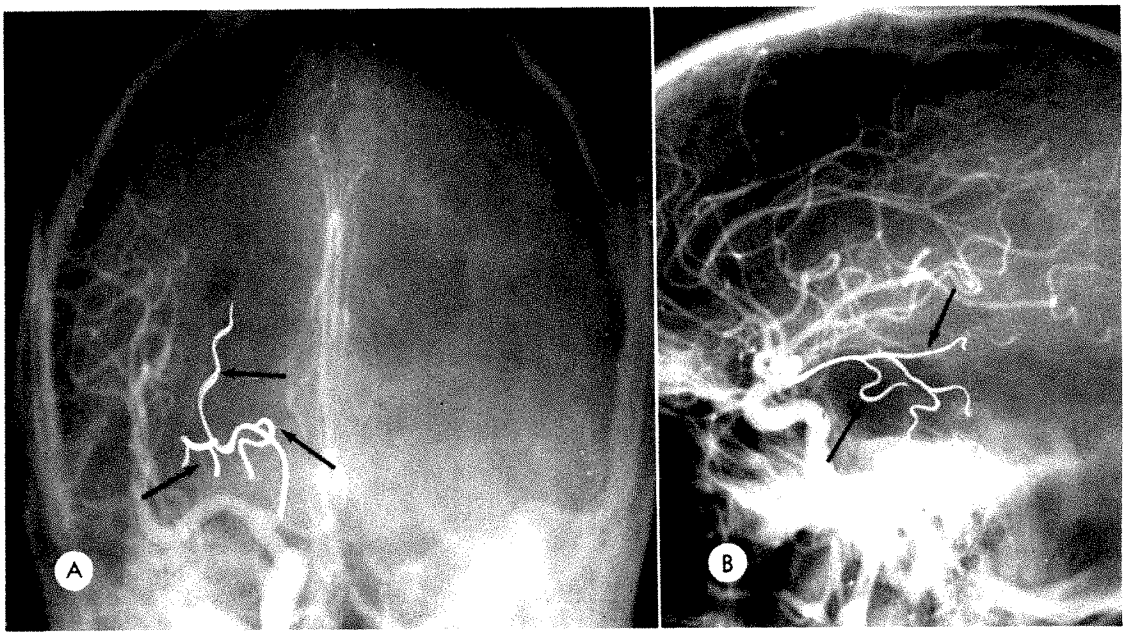

Fig. 14. (*A* and *B*) Large branches from the anterior choroidal artery in a 34 year old man suggested basilar-artery disease which was confirmed arteriographically.

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UNILATERAL INTRACRANIAL HEMATOMA WITHOUT SHIFT OF THE ANTERIOR CEREBRAL ARTERY

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DISPLACEMENT of the anterior cerebral artery across the midline is generally accepted as suggestive evidence of a space-occupying intracranial lesion. However, a normal arteriogram on one side does not necessarily exclude a mass lesion on the opposite side although this has been implied in some articles, particularly those concerned with intracranial hematoma.[3,7] Reports have been presented which indicate that the anterior cerebral artery may remain in or very near the midline despite the presence of a sizable unilateral space-occupying lesion.[1,4,5] This finding is occasionally associated with hematomas in the subdural space or temporal lobe, and bilateral carotid arteriography is therefore frequently necessary when such a lesion is suspected. Arteriography has admitted defects; it is an unpleasant experience for the patient, and the complications, although infrequent, may be severe. Echoencephalography has been proposed as a safe, painless, and effective alternative procedure. In a recent report the statement is made that patients with head injury were "treated or left alone on the basis of the echoencephalogram."[2] The echoencephalographic findings

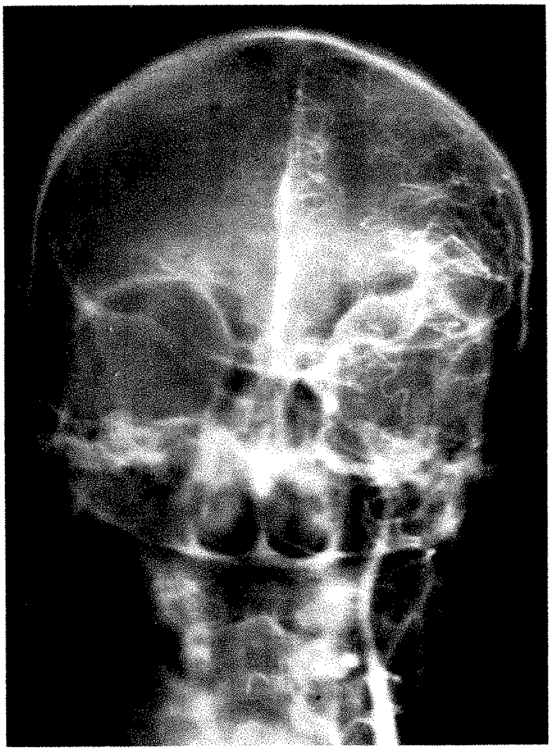

Fig. 1. Case 1. Anteroposterior projection. Intracerebral hematoma of the left temporal lobe. There is elevation and medial displacement of the middle cerebral artery without shift of the anterior cerebral artery.

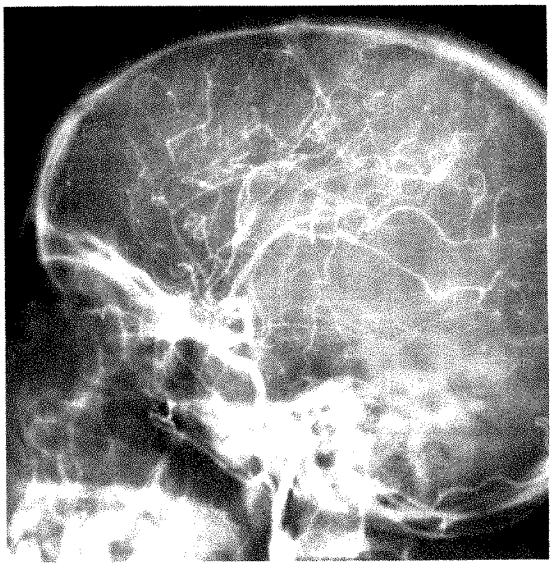

Fig. 2. Case 1. Left lateral projection. The middle cerebral artery is elevated. The arteriogram of the right side was normal.

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considered included the detection of echoes at the hematoma-brain interface and difficulty in obtaining echoes on the side of the subdural hematomas. However, displacement of midline structures was the most consistent and frequently encountered abnormality and appeared to be the most important factor in determining whether further measures were to be undertaken.

MATERIAL

During the past 3 years we have observed 15 cases of unilateral post-traumatic intracranial hematoma without an arteriographically demonstrable shift of the anterior cerebral artery. A number of additional cases where the shift was equivocal and the determination complicated by rotation of the head have not been included. Either bilateral arteriograms or bilateral

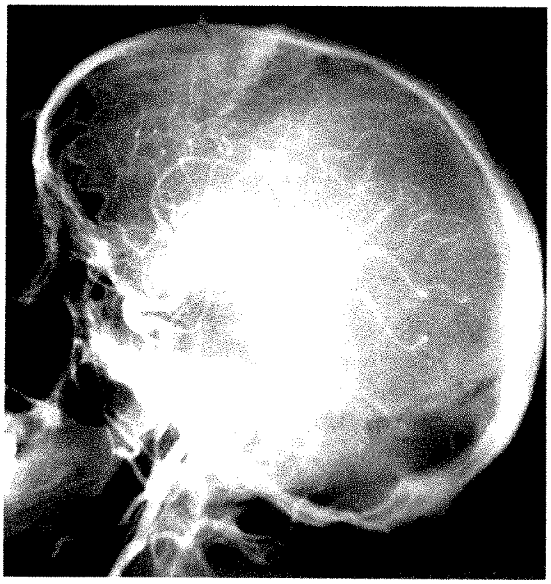
Fig. 4. Case II. Left lateral projection. The middle cerebral artery is elevated.

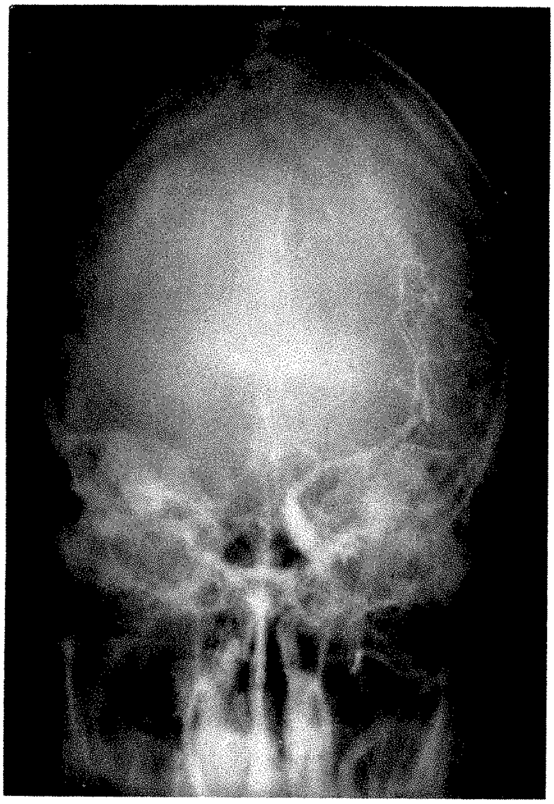
Fig. 3. Case II. Anteroposterior projection. Intracerebral hematoma of the left temporal lobe. The middle cerebral artery is elevated, while the anterior cerebral artery is slightly deviated toward the left.

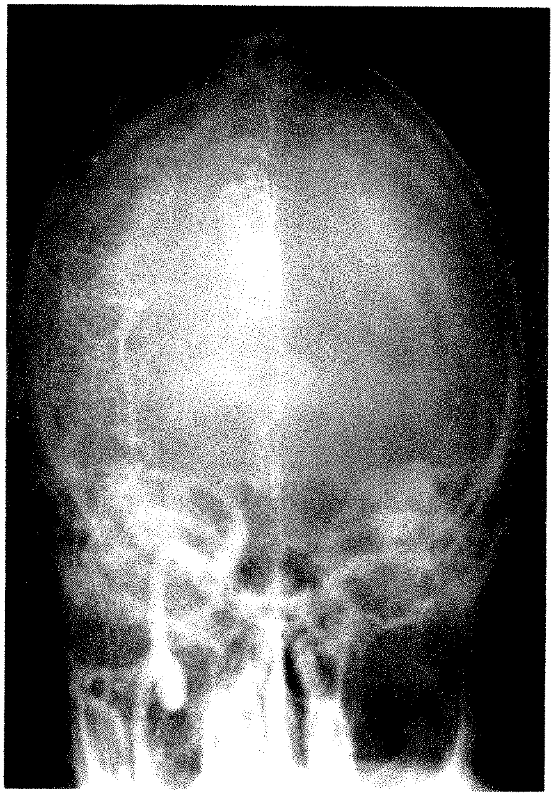
Fig. 5. Case II. Arteriogram showing normal configuration of the right middle cerebral artery.

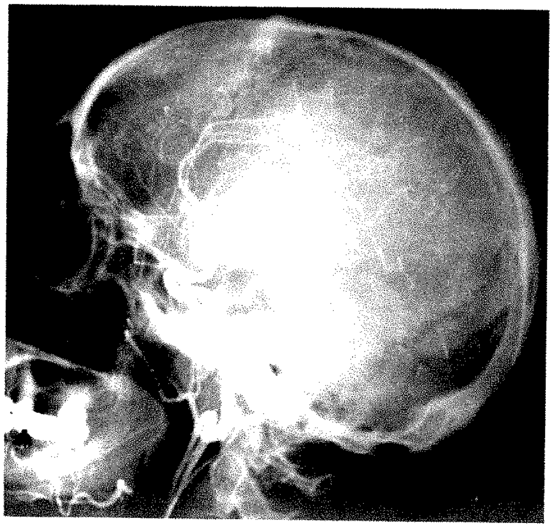

Fig. 6. Case II. Normal right lateral arteriogram.

burr holes were done in all cases; in some instances, both were done.

ROENTGEN FINDINGS

In all cases the anterior cerebral artery remained in the midline despite the presence of sizable unilateral hematomas. Eleven of the lesions were temporal lobe intracerebral hematomas; 4 were subdural hematomas. The internal cerebral vein was identified infrequently, in 7 of the cases because of single exposure techniques. All procedures were done under local anesthesia. The patients were often restless and disoriented, and when serial studies were done, difficulty was encountered in preventing rotation of the head during the venous phase. However the internal cerebral vein was undisplaced in 2 of the 3 cases where it was adequately demonstrated.

In patients with definite lateralizing findings or progressive depression of consciousness, a normal unilateral study may not as readily be taken as indicating a normal state on the opposite side. In those individuals with only mental abnormalities, the diagnosis of a mass lesion is less likely to be made if the midline structures have

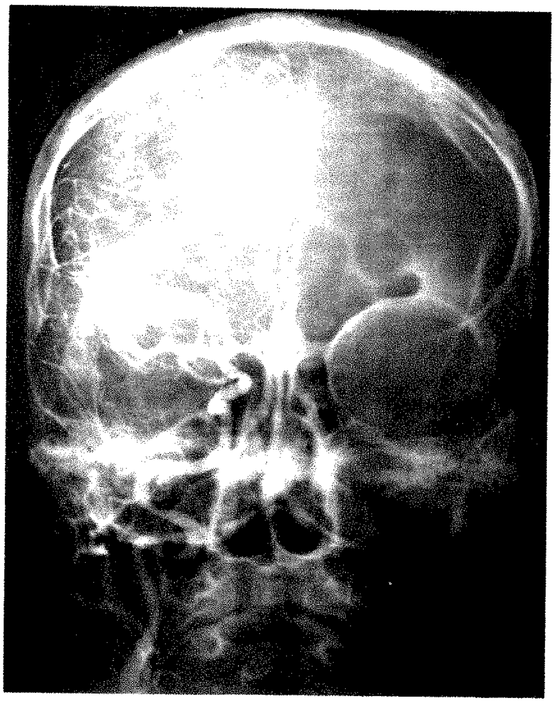

Fig. 7. Case III. Right carotid arteriogram interpreted as normal.

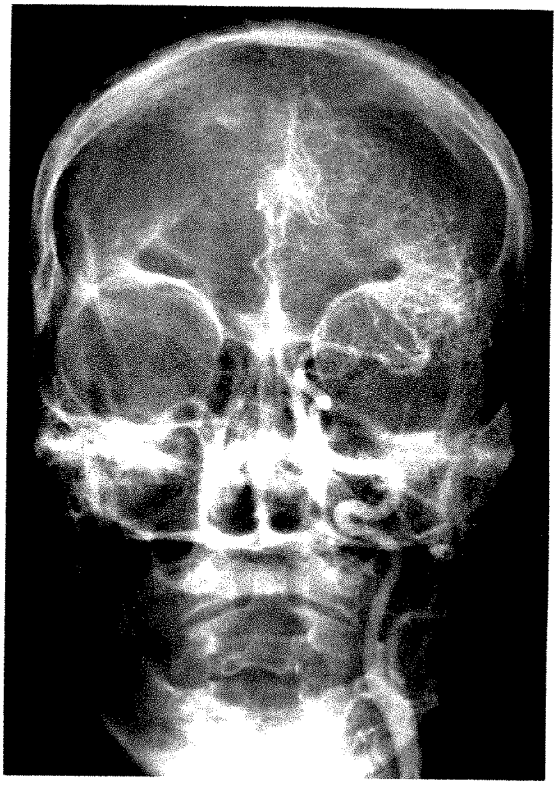

Fig. 8. Case III. Left carotid arteriogram showing a large subdural hematoma. The rotation to the right produces an apparent shift of the anterior cerebral artery.

been shown to be undisplaced by a single diagnostic procedure.

The following case reports illustrate the unreliability of failure of shift of the anterior cerebral artery across the midline in the diagnosis of unilateral intracranial hematoma.

REPORT OF CASES

Case i. This 43 year old woman was admitted to the hospital 2 days after having been beaten. She was grossly disoriented, noisy, and agitated. Although the patient had occasional brief intervals of relatively normal behavior, sustained improvement did not occur. Repeated examinations failed to disclose any other neurologic abnormalities. Bilateral carotid arteriograms were made 4 days after admission. The right arteriogram was normal, the left was compatible with a temporal lobe lesion (Fig. 1 and 2). The patient was operated upon and an intracerebral hematoma was evacuated from the left temporal lobe. Following surgery, the patient improved rapidly and was well oriented and apparently normal when discharged on the 12th postoperative day. She returned to work and was able to function satisfactorily.

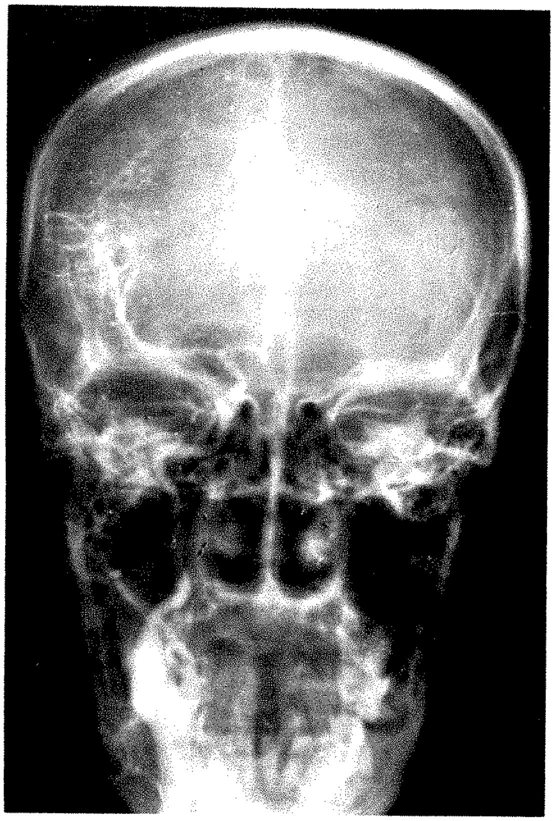

Fig. 9. Case iv. Intracerebral hematoma of the right temporal lobe. The middle cerebral artery is slightly displaced superiorly.

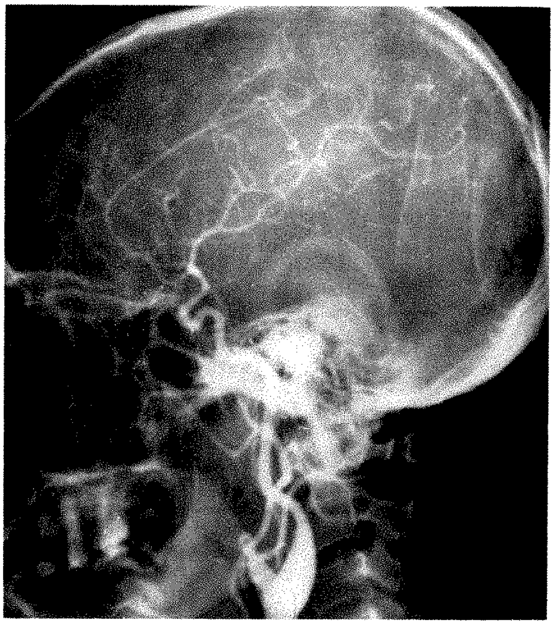

Fig. 10. Case iv. In the lateral projection, the middle cerebral artery is elevated. The arteriogram of the left side was normal.

Case ii. A 38 year old man was admitted to the hospital after a head injury. Briefly unconscious initially, he was alert and responsive when first seen in the admitting room, then became restless and very disoriented. He talked incessantly but not coherently. His repeated attempts to leave the hospital made the use of restraints necessary. Hallucinations were frequent. The patient's condition remained unchanged and lateralizing findings did not appear. Bilateral carotid arteriograms were made 1 week after injury (Fig. 3, 4, 5 and 6). The abnormalities were limited to the left side and were interpreted as demonstrating a temporal lobe lesion. He was operated upon and a large temporal intracerebral hematoma was removed. The postoperative course was good, with prompt and progressive recovery.

Case iii. This 56 year old man had been found wandering in the streets and was brought to the hospital by the police. He was alert but confused. The neurologic examination was

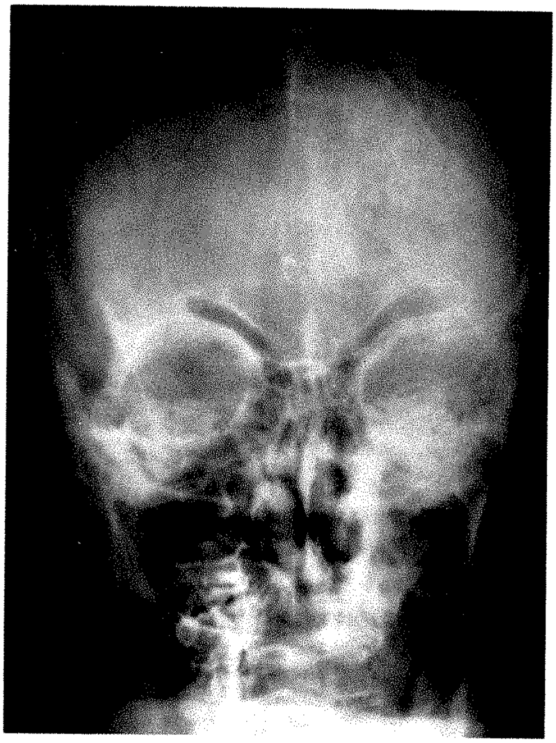

Fig. 11. Chronic left subdural hematoma without displacement of the internal cerebral vein.

otherwise unremarkable. A right carotid arteriogram made several days later was interpreted as normal (Fig. 7). The left carotid arteriogram, however, demonstrated a large subdural hematoma (Fig. 8). The hematoma was drained surgically. The patient improved steadily after the operation and appeared normal when released from the hospital.

Case iv. This 33 year old man had been hit on the head during a brawl and was unconscious for a short time thereafter. He was agitated, noisy, and restless, and could not give a coherent history. His speech was rambling and disjointed. Other neurologic abnormalities were not observed. The patient's behavior remained unchanged. Bilateral carotid arteriograms made 8 days after injury were suggestive of a right temporal lobe mass (Fig. 9 and 10), with normal findings on the left side. An intracerebral hematoma was removed from the right temporal lobe. The postoperative course was uncomplicated and the patient made a good recovery.

DISCUSSION

The occurrence of unilateral intracranial hematomas without shift of the anterior cerebral artery indicates the necessity for bilateral arteriograms in some patients and implies that diagnostic procedures which rely, to a great extent, upon the determination of midline structures may not be adequate to exclude a space-occupying lesion. It is true that structures more centrally located may sometimes be displaced while the anterior cerebral artery is not. In 2 of our cases, however, the internal cerebral veins were not shifted (Fig. 11 and 12), suggesting that the third ventricle also remained in the midline. If the anterior cerebral artery above and the internal cerebral veins below are undisplaced, it is unlikely that the septum pellucidum could be significantly deviated.

CONCLUSIONS AND SUMMARY

Fifteen cases of unilateral post-traumatic intracranial hematomas without arteriographic evidence of anterior cerebral artery displacement are reported. The findings indicate that a normal unilateral carotid ar-

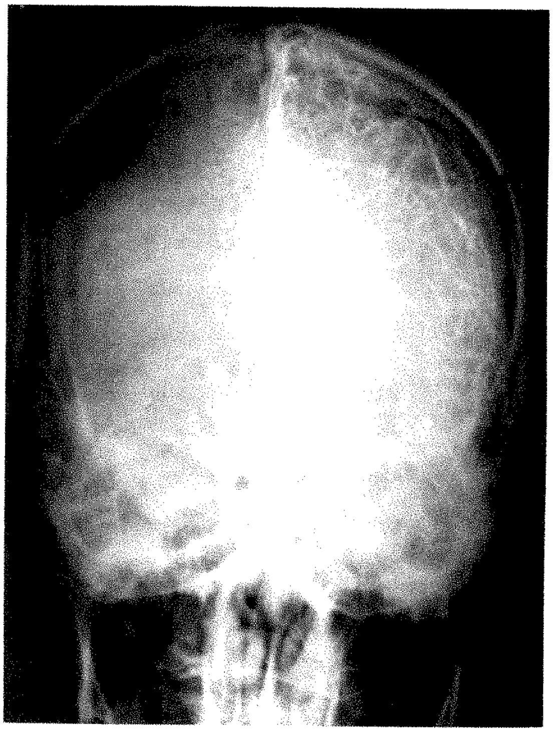

Fig. 12. Parieto-occipital extradural hematoma without a shift of the internal cerebral vein.

teriogram does not constitute an adequate roentgenologic examination when an intracranial hematoma is suspected. Similarly, identification of undisplaced midline structures is insufficient evidence to exclude a space-occupying lesion.

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ANGIOGRAPHIC FEATURES OF UNILATERAL HYDROCEPHALUS OF OBSTRUCTIVE NATURE*

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THE presence of an expanding mass lesion in a cerebral hemisphere is immediately suspected when mid-line structures are displaced to the opposite side of the cranial cavity. The major mid-line structures are the pineal gland, the septum pellucidum and third ventricle, and the anterior cerebral artery and internal cerebral vein. In some cases, however, contralateral displacement is not the direct result of the mass but is secondary to obstruction by the mass of the lateral ventricle with resulting marked dilatation of this ventricle under pressure. Recognition that displacement is caused by a large lateral ventricle is obvious on pneumoencephalography or ventriculography when the involved ventricle is filled with gas. However, on carotid angiography, the suspicion that displacement is the result of unilateral hydrocephalus may not arise unless this possibility is kept in mind and special attention is paid to the configuration of the veins in the walls of the ventricle. The distinction between displacement by an expanding mass *per se* and displacement due to unilateral obstructive hydrocephalus is of importance since, in the latter case, relief of the obstruction by surgical intervention may result in substantial palliation.

Obstructive hydrocephalus of one lateral ventricle, that is, unilateral hydrocephalus, may be divided into two types. The first type is the result of obstruction at one of the foramina of Monro which results in generalized or uniform dilatation of the lateral ventricle. This is usually the result of a neoplasm in the region of the foramen which may arise either in adjacent portions of the lateral ventricle or in the third ventricle. Gliomas including ependymomas, colloid cysts and intraventricular craniopharyngiomas are the most common neoplasms in this area. In the second type, the obstruction is within the lateral ventricle and only a portion of the ventricular system is dilated as a result of trapping of ventricular fluid proximal to the site of obstruction. Neoplasms causing this type of partial unilateral hydrocephalus include those that are primarily intraventricular, such as meningiomas, ependymomas, papillomas and cysts of the choroid plexus, and those that arise in the wall of the ventricle and secondarily protrude into the ventricular cavity. This last group may be referred to as paraventricular tumors.

The angiographic features of unilateral obstructive hydrocephalus include findings due to shift of mid-line structures to the opposite side and findings resulting from dilatation of the lateral ventricle. The degree of shift of the anterior cerebral or rather pericallosal artery is less than that of the internal cerebral vein but this is a nonspecific feature indicating simply that the lesion is not likely to be located in the frontal lobe. The contralateral displacement of the pericallosal artery is uniform without marked convexity toward the opposite side and is the result of herniation underneath the falx. The pericallosal artery shows a wide sweeping or bowed course around the genu and elongation or a windblown appearance of its cortical branches. The insular portions of the middle cerebral artery are displaced laterally and the temporal and parietal branches are stretched and separated from each other. The posterior cerebral artery may be displaced downward and its branches elongated. The posterior callosal or splenial artery, a branch of the posterior cerebral artery, is elevated. The anterior choroidal artery is displaced medially. The posterior choroidal arteries run more anteriorly than normally as they extend upward in the anterior wall of the atrium. The terminal portions of these vessels in the velum interpositum and floor of the lateral ventricle may be displaced downward. The striate branches of the anterior and middle cerebral arteries as seen in the anteroposterior projection show a more markedly inclined or tilted course as they run upward and laterally. The negative findings in the arterial and capillary phases are also of significance, specifically the lack of any localized deformity or displacement of the arteries on the surface of the brain and the lack of any stain related to the cortical portions of the hemisphere. In other words, no specific localizing evidence is present although a mass lesion is suspected. The temptation to attribute stretching and elongation of arterial branches to a diffusely infiltrating neoplasm must be resisted. This error is most likely to occur when dilatation is confined to one portion of the ventricle such as the atrium or temporal horn.

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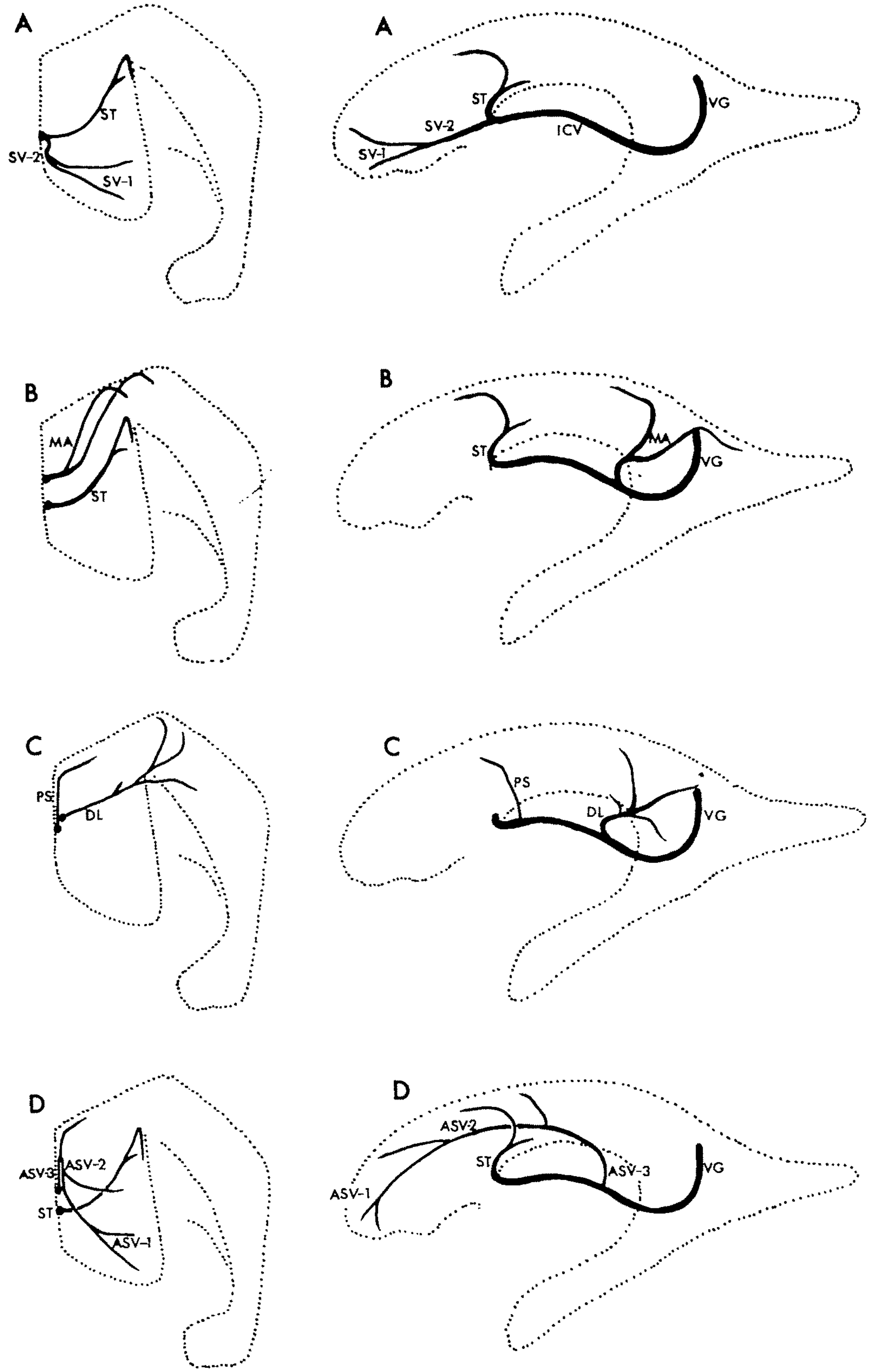
The changes in the internal cerebral vein and the striothalamic or posterior terminal vein in cases of marked dilatation of the lateral ventricle have been described by several authors.[1-5] The internal cerebral vein is flattened and may appear not to extend as far forward as normally because of widening of the foramen of Monro. The flattening of the internal cerebral vein extends into the great vein of Galen without any demarcation. The internal cerebral vein may also be displaced downward particularly if the third ventricle is of normal size. The venous angle or junction between the striothalamic vein and the internal cerebral vein is low and widened in both the lateral and anteroposterior projections. The striothalamic vein is displaced laterally and elongated and its tributaries arise unusually high. The curved course of this vein in the floor of the lateral ventricle is exaggerated and unusually low. A substantial amount of additional information can be obtained from other less well-known subependymal veins. Study of these vessels is of particular importance when the lateral ventricle is not uniformly dilated since they assist in identifying portions of the ventricular walls not related to the striothalamic vein. A classification of subependymal veins into medial and lateral groups corresponding to the medial and lateral walls of the ventricle has been suggested.[6] This classification is useful in determining ventricular size since any increase in the transverse distance between veins of the medial and lateral groups must indicate an increase in the size of the ventricle itself. In unilateral obstructive hydrocephalus, veins of the medial group are displaced medially or to the opposite side while veins of the lateral group are displaced laterally. The medial group of veins includes the vein of the septum pellucidum, posterior septal veins, the medial atrial veins ("vein of the posterior horn") and small veins in the floor of the temporal horn. The lateral group includes anterior caudate veins, the striothalamic vein, the direct lateral vein, the lateral atrial vein and the inferior ventricular vein. The course of these veins and common variations have been previously described.[6] The appearance of these vessels is sufficiently characteristic so that they can usually be identified in the normal (Fig. 1, *A–D*) or the dilated ventricle (Fig. 2, *A–D*). With dilatation, the subependymal veins are stretched, elongated and separated from each other with exaggerated concavities toward the lumen of the ventricle. The most common variation of significance in the present context is the presence of a long septal vein which runs for a considerable distance posteriorly within the septum pellucidum and then turns downward to drain into the middle or posterior portion of the internal cerebral vein (Fig. 1*D* and 2*D*). The presence of such an anomalous septal vein is fortunate since it serves to identify the medial wall of the ventricle behind the foramen of Monro.

The features of ventricular dilatation described above for unilateral hydrocephalus are essentially the same as those seen in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bilateral dilatation except for contralateral displacement of mid-line structures. However, the degree of dilatation in unilateral obstructive hydrocephalus is often considerably more marked, partly due to the fact that the opposite ventricle is not enlarged. If bilateral angiography is performed, there will be an obvious difference in the appearance of the subependymal veins when the ventricles are unequal in size. The internal cerebral vein on the unobstructed side is at a higher level and has a more normal sinuous course. The striothalamic vein of the uninvolved side is smaller and closer to the mid-line. It is of interest that the pericallosal arteries are usually symmetric even though dilatation is confined to one lateral ventricle. An exaggerated sweep or elevation of these arteries may not be present when the corpus callosum is oblique and not elevated on the contralateral or nonobstructed side toward which these vessels are displaced.

While the general principles basic to the understanding of the angiographic changes in unilateral obstructive hydrocephalus are clear-cut, their application to specific cases may be surprisingly difficult. Problems arise in recognizing the various subependymal veins and in interpreting the venous phases when these vessels are markedly displaced or anomalous. The anteroposterior projection is particularly confusing because of overlapping vessels. These difficulties in interpretation are best illustrated by examining individual cases in detail.

REPORT OF CASES

CASE I. A 30 year old housewife was admitted March 1, 1957 with chief complaints of progressive headache, unsteady gait and mental confusion of 7 weeks' duration. Frequent vomiting had been present for about 3 weeks. On examination, she was found to have bilateral papilledema, right facial weakness, a right Babinski reflex and increased deep tendon reflexes on the right side. The patient's ability to calculate was obviously diminished and she showed paranoid ideas. Spinal tap showed a cerebrospinal fluid pressure of 580 mm. of water. The total protein of the spinal fluid was 30 mg. per cent. Left carotid angiograms (Fig. 3, *A*, *B*, *C* and *D*) were made shortly after admission and suggested a deep mass near the foramen of Monro. A left frontal craniotomy was performed on March 6, 1957. This revealed an infiltrating tumor in the posterior portion of the septum pellucidum obstructing the foramen of Monro on the left side. The left lateral ventricle was markedly dilated. Subtotal resection of the tumor was performed. In addition, an opening was made in the septum pellucidum behind the foramen of Monro in order to establish free communication between the two lateral ventricles. The histo-

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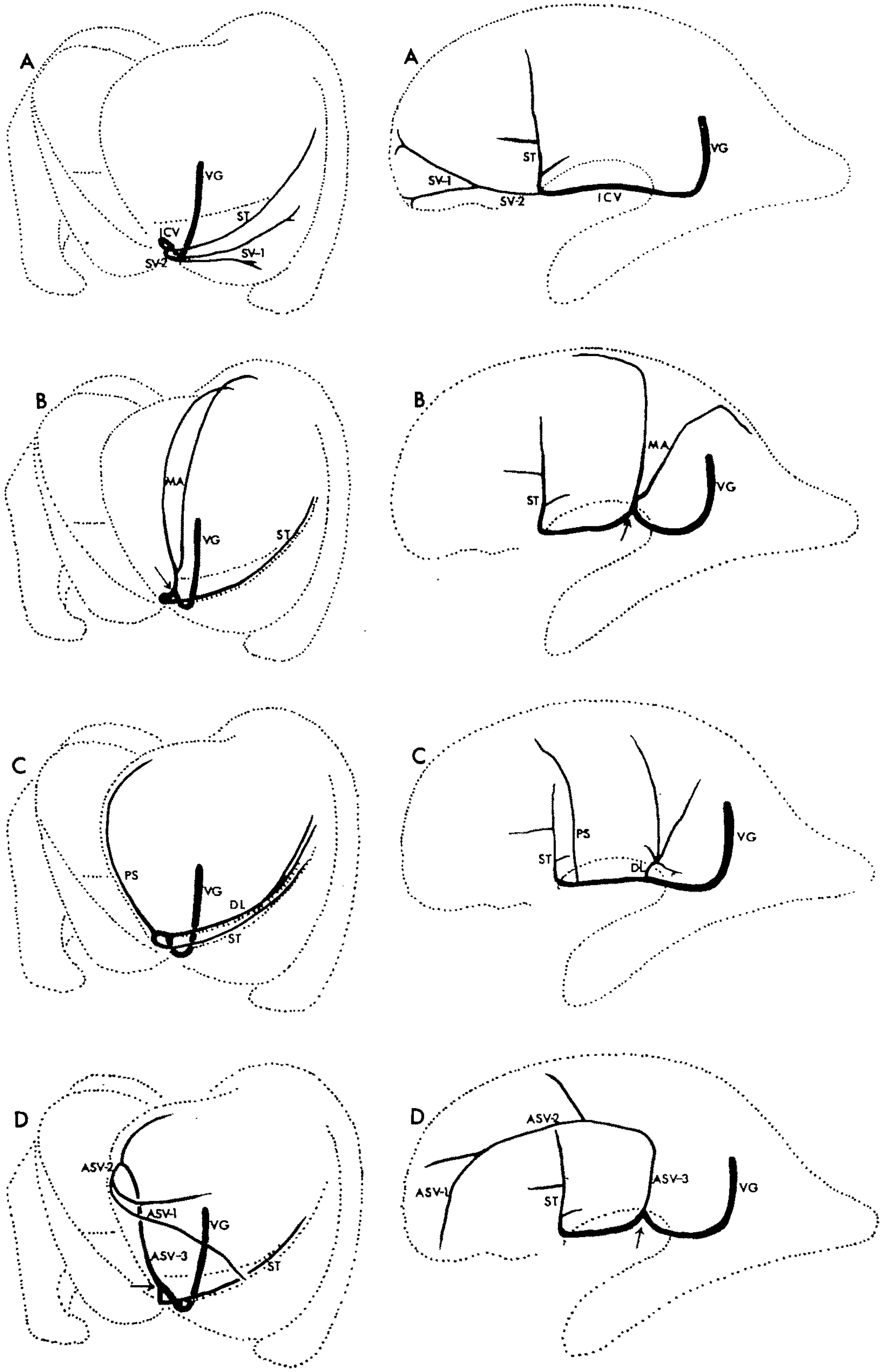
FIG. 1. Diagrammatic representation of normal subependymal veins draining into the internal cerebral vein. Veins of importance in determining the size of the ventricle are illustrated.

(*A*) Anteroposterior and lateral view. The striothalamic vein (ST) is located in the lateral wall and floor of the lateral ventricle. The septal veins are located in the anterior (SV-1) and medial (SV-2) walls of the frontal horn. The internal cerebral vein (ICV) is shown only in the lateral view. The vein of Galen is labeled.

(*B*) Anteroposterior and lateral view. The medial atrial veins (MA) are located in the medial wall of the posterior portion of the body, atrium and posterior horn. The striothalamic veins are also illustrated.

(*C*) Anteroposterior and lateral view. The direct lateral veins (DL) are located in the floor and lateral wall of the body of the lateral ventricle and anterior wall of the atrium. A posterior septal vein (PS) is located in the roof of the body of the ventricle and in the septum pellucidum behind the foramen of Monro.

(*D*) Anteroposterior and lateral view. A large anomalous septal vein begins in the anterior wall of the frontal horn (ASV-1), runs in the upper portion of the septum pellucidum (ASV-2), and turns downward in the posterior end of the septum (ASV-3) to join the internal cerebral vein. Tributaries from the roof join the horizontal portion of this vein.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logic report was astrocytoma Grade I. The postoperative course was excellent. The neurologic signs regressed to a considerable extent and she was discharged in 2 weeks under the care of the Radiotherapy Department. Following completion of radiation therapy, she did well for about 6 months but then developed increasing mental confusion and progressive right hemiparesis. She improved again, however, after further radiation therapy and is doing surprisingly well 6 years after operation.

Comment. Carotid angiograms on this patient showed the presence of unilateral hydrocephalus rather clearly. The possibility of a mass near the foramen of Monro was suggested on the basis of posterior displacement of the foramen of Monro as well as the angulation and somewhat irregular configuration of the veins at the junction of the striothalamic stem and the internal cerebral vein.

Case II. An 8 year old boy was admitted on December 13, 1957 with chief complaints of headache and diminished vision of the left eye of short duration. Neurologic examination showed evidence of diminished visual acuity on both sides, more marked on the left. There was increased intracranial pressure as indicated by enlargement of the head, a McEwen sign, and bilateral papilledema. Spinal puncture showed cerebrospinal fluid pressure of 280 mm. of water. Cerebrospinal fluid contained 129 mg. per cent of protein. Plain roentgenograms of the skull showed separation of the cranial sutures. Pneumoencephalogram showed filling only of the right lateral ventricle which was slightly enlarged and displaced towards the right. No air entered the left lateral ventricle. Left carotid angiograms (Fig. 4, *A*, *B*, *C* and *D*) were interpreted as indicating marked enlargement of the left lateral ventricle probably due to obstruction in the region of the foramen of Monro. Pantopaque ventriculography was attempted but the opaque material entered the subdural space. This was followed by pneumoventriculography (Fig. 4, *E* and *F*) which revealed a mass in the left lateral ventricle in the region of the foramen of Monro. Through a left frontal craniotomy, the frontal horn was opened and a tumor was found apparently arising from the medial portion of the floor of the lateral ventricle adjacent to the foramen of Monro. The left lateral ventricle was markedly dilated. A subtotal removal of the tumor was performed and an opening in the septum pellucidum made to permit free communication between the lat-

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Fig. 2. Diagrammatic representation of the subependymal veins in unilateral hydrocephalus. The major vessels shown in Figure 1 are illustrated as they appear in marked dilatation of the lateral ventricle.

(*A*) Anteroposterior and lateral view. The internal cerebral vein (ICV) is displaced to the opposite side, depressed and flattened. The striothalamic vein (ST) is displaced laterally and downward and shows a wide sweeping curved course. The tributaries of the septal vein in the anterior wall (SV-1) begin unusually far anteriorly and laterally. The septal portion of this vein (SV-2) is displaced to the opposite side. The great vein of Galen (VG) is also labeled.

(*B*) Anteroposterior and lateral view. The medial atrial vessels (MA) are markedly elongated and medially displaced. The internal cerebral vein is kinked upward (arrow) at the entrance of the medial atrial vein.

(*C*) Anteroposterior and lateral view. The direct lateral vein (DL) is markedly elongated and begins unusually far laterally. In the anteroposterior projection, the course of this vessel resembles the striothalamic vein (ST) but does not extend as far inferiorly. A posterior septal vein (PS) outlines the medially displaced septum pellucidum behind the foramen of Monro. In the lateral projection, the course of this septal vessel simulates the striothalamic vein.

(*D*) Anteroposterior and lateral view. A large elongated anomalous septal vein outlines the anterior wall (ASV-1) of the frontal horn, runs along the upper margin of the septum (ASV-2) and then down to the displaced, stretched posterior portion of the septum (ASV-3). The internal cerebral vein is kinked upward (arrow) at the entrance of the septal vein.

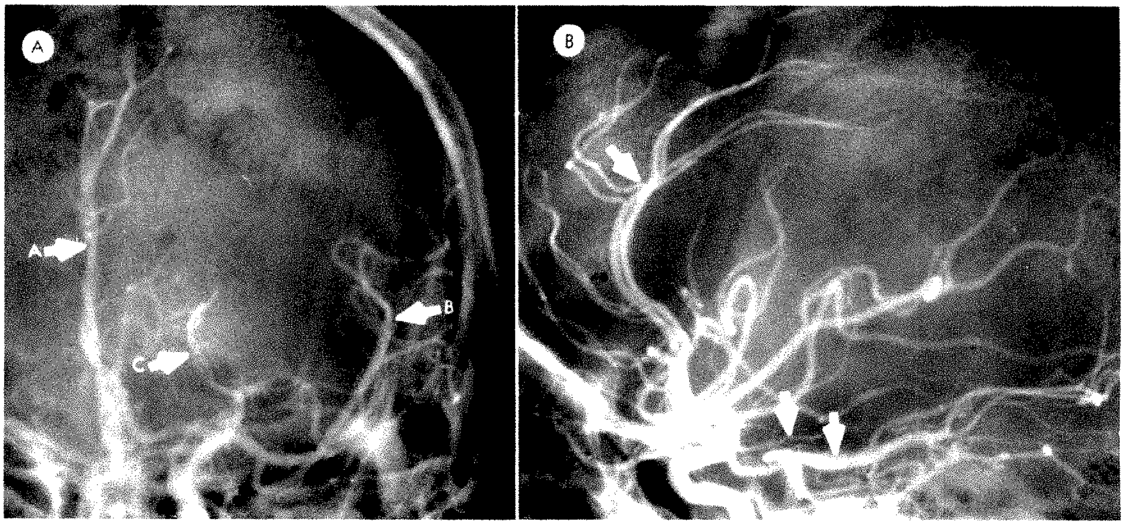

Fig. 3. Case i. Astrocytoma (Grade i) of the septum pellucidum with occlusion of the ipsilateral foramen of Monro.

(*A*) Arterial phase—anteroposterior view. The pericallosal artery and its branches (arrow A) are displaced toward the opposite, *i.e.*, right side. The insular portion of the middle cerebral artery (arrow B) is displaced slightly laterally. The posterior cerebral artery (arrow C) is not remarkable.

(*B*) Arterial phase—lateral view. Both pericallosal arteries (upper arrow) are filled and show an identical wide sweeping course. The anterior choroidal and the posterior cerebral arteries (lower arrows) are stretched and flattened. The middle cerebral arteries are stretched but not displaced.

eral ventricles. The histologic diagnosis was astroblastoma. The postoperative course was smooth. Radiation therapy was administered and the patient was discharged. The patient was lost to further follow-up.

Comment. At the time of carotid angiography, the individual subependymal veins were not separately identified. Nevertheless, the possibility of a deep lesion near the foramen of Monro was suggested on the basis of secondary signs and by exclusion. In retrospect, the irregular, markedly depressed course of the striothalamic stem suggests the actual site of the neoplasm.

Case iii. This was the first admission of a 57 year old white female with chief complaints of blurring of vision, difficulty in balancing herself, defective memory for recent events and easy fatigability for about 4 months. In addition for the same period of time, she also noted that she drank excessively and was quite confused particularly on awakening in the morning. For a period of 2 or 3 weeks, she complained of moderately severe bifrontal headache. Neurologic examination showed marked ataxia with a positive Romberg sign, a slight right facial palsy and an organic mental syndrome. There was also some evidence of a right homonymous hemianopsia but only on double simultaneous stimulation. Electroencephalography showed some abnormality on the left side which was most marked in the temporal area. Left carotid angiograms (Fig. 5, *A*, *B*, *C* and *D*) showed evidence of dilatation of the left lateral ventricle with a shift of the mid-line structures to the opposite side. Although a specific diagnosis was not made, the suspicion was raised of occlusion of the foramen of Monro on the left side. Pneumoencephalograms (Fig. 5, *E* and *F*) demonstrated a sharply demarcated filling defect in the anterior inferior portion of the third ventricle. A left frontal craniotomy was performed. Through a transcortical incision, the right frontal horn was entered and a bulging greenish mass was seen protruding through the foramen of Monro from the third ventricle. Further exposure was obtained by splitting the fornix. Five cubic centimeters of greenish fluid was aspirated from the mass. The tumor was then excised, presumably completely. The mass appeared to be attached to the anterior inferior portion of the third ventricle. The histologic diagnosis was craniopharyngioma. Postoperatively, the condition of the patient was precari-

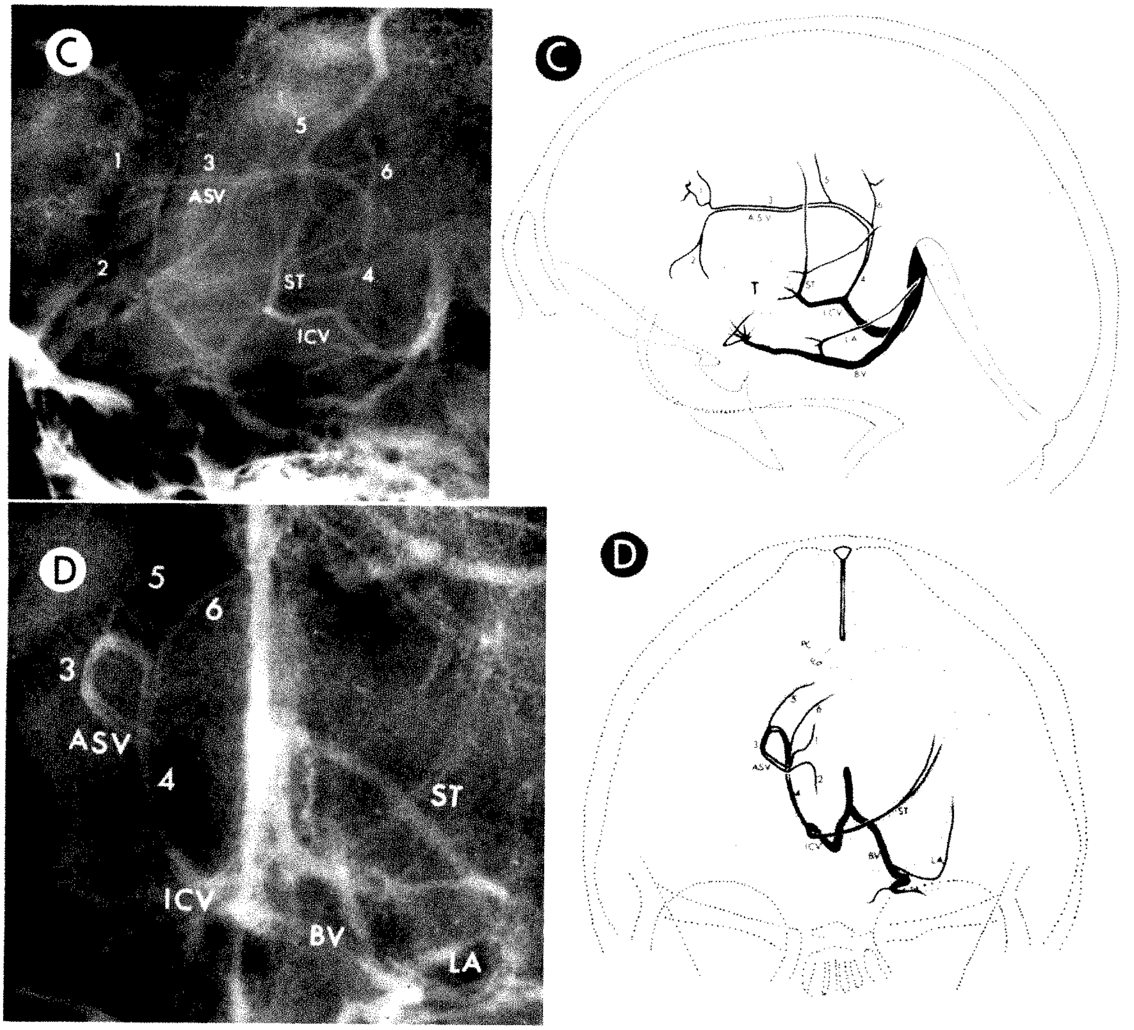

Fig. 3. (*C*) Venous phase—lateral view and line drawing of it. An anomalous double-channeled septal vein (ASV) enters the internal cerebral vein (ICV) near its mid-portion. One channel (1) begins in the superior portion of the frontal horn while the other (2) originates in the inferior portion. The double channel (3) runs posteriorly in the upper part of the stretched septum pellucidum and then turns rather abruptly downward (4) to enter the internal cerebral vein at a right angle. Several tributaries (5 and 6) from the roof of the lateral ventricle join the main channel. The junction with the internal cerebral vein is kinked upward. The striothalamic stem (ST) and its tributaries are markedly elongated in a vertical direction. The junction of the striothalamic vein and the internal cerebral vein (foramen of Monro) is displaced posteriorly. The internal cerebral vein is depressed downward and its anterior portion shows a zig-zag course. A lateral atrial vein (LA) in the anterolateral wall of the atrium and roof of the temporal horn is clearly seen and assists in visualizing this portion of the ventricular system. The basal vein (BV) is displaced slightly downward. The tumor found at operation (T) is indicated in shaded fashion.

(*D*) Venous phase—anteroposterior view and line drawing of it. The internal cerebral vein (ICV) is displaced toward the right side. The anomalous septal vein (ASV) is markedly displaced medially. The tributaries (1 and 2) of this vein in the anterior wall are deviated less than the septal portions of the vessel (3 and 4). The roof tributaries (5 and 6) show a smoothly curved course as they enter the septum pellucidum to join the anomalous septal vein. The normally distinct angle between the roof and the medial wall is effaced. The striothalamic vein (ST) and its tributaries have a stretched arcuate course as they run in the lateral wall and floor of the lateral ventricle. These vessels are displaced markedly laterally and downward. The curvilinear course and separation of the striothalamic and septal veins and their tributaries indicate marked enlargement of the lateral ventricle in all directions. The lateral atrial vein (LA) and the basal vein (BV) can also be identified. The left lateral ventricle is shown in shaded fashion. (The location of the pericallosal cistern (PC) in the diagram is based upon the arterial phase.)

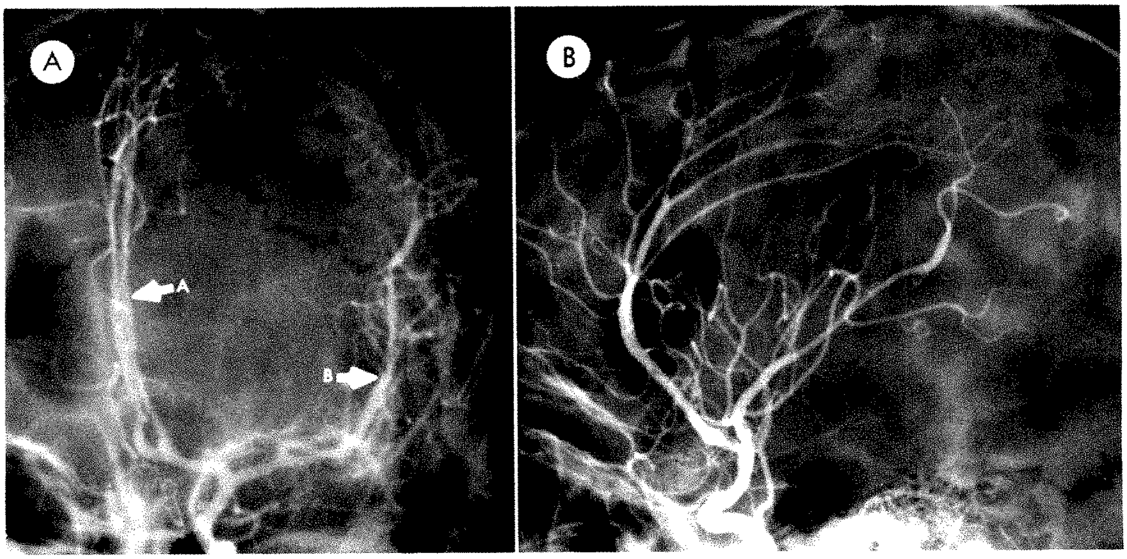

Fig. 4. Case II. Astroblastoma in the region of the left foramen of Monro extending into the lateral and third ventricles.

(*A*) Arterial phase—anteroposterior view. Both pericallosal arteries (arrow A) are filled and displaced toward the right. The middle cerebral artery (arrow B) is displaced laterally. (The small amount of air in the right frontal horn was the result of previous pneumoencephalography which failed to visualize the left lateral ventricle.)

(*B*) Arterial phase—lateral view. The pericallosal arteries show a wide sweeping course. Apparent elevation of the middle cerebral artery is due to rotation of the head as evidenced by unequal heights of the orbital roofs.

ous and because of increased intracranial pressure and progressive drowsiness, a ventriculoatrial shunt was performed 48 hours after the first operation. The patient, however, responded to this procedure poorly and expired 11 days after the original operation. No autopsy was obtained.

Comment. In this patient, the elevation of the anterior end of the internal cerebral vein in spite of the marked dilatation of the lateral ventricle suggested enlargement of the third ventricle. However, since there was a mid-line shift of the ventricular system, this is not likely to be the result of simple dilatation of the third ventricle but rather of pressure by the mass within it. This was further confirmed by lateral displacement of the anterior end of the basal vein.

Case IV. This was the first admission of an 11 year old boy with a history of right sided seizures for about 4 years. These became more frequent in the previous 4 months. The child also experienced occasionally mild headache. Shortly before admission, pneumoencephalography had been performed but the results were not available. Examination on admission showed only some slight weakness of the right hand and increased deep tendon reflexes on the right side. Plain roentgenograms of the skull showed the left side to be considerably larger than the right and the calvaria on the left was thinner than on the right. Left carotid angiograms (Fig. 6, *A*, *B*, *C* and *D*) were interpreted as indicating the presence of a large intra-atrial mass. This was later confirmed by pneumoencephalograms (Fig. 6, *E* and *F*). At operation, a large cyst was removed from the left lateral ventricle following incision of the markedly thinned parietal cortex. The cyst was attached to the medial wall of the atrium and contained clear fluid. Histologic examination of the wall of the cyst showed only connective tissue but the findings were nevertheless considered to be compatible with an ependymal cyst. Portions of the membrane, however, showed fibers that resembled those seen in the arachnoid. He was discharged, markedly improved 2, weeks after operation.

Comment. The angiographic study in

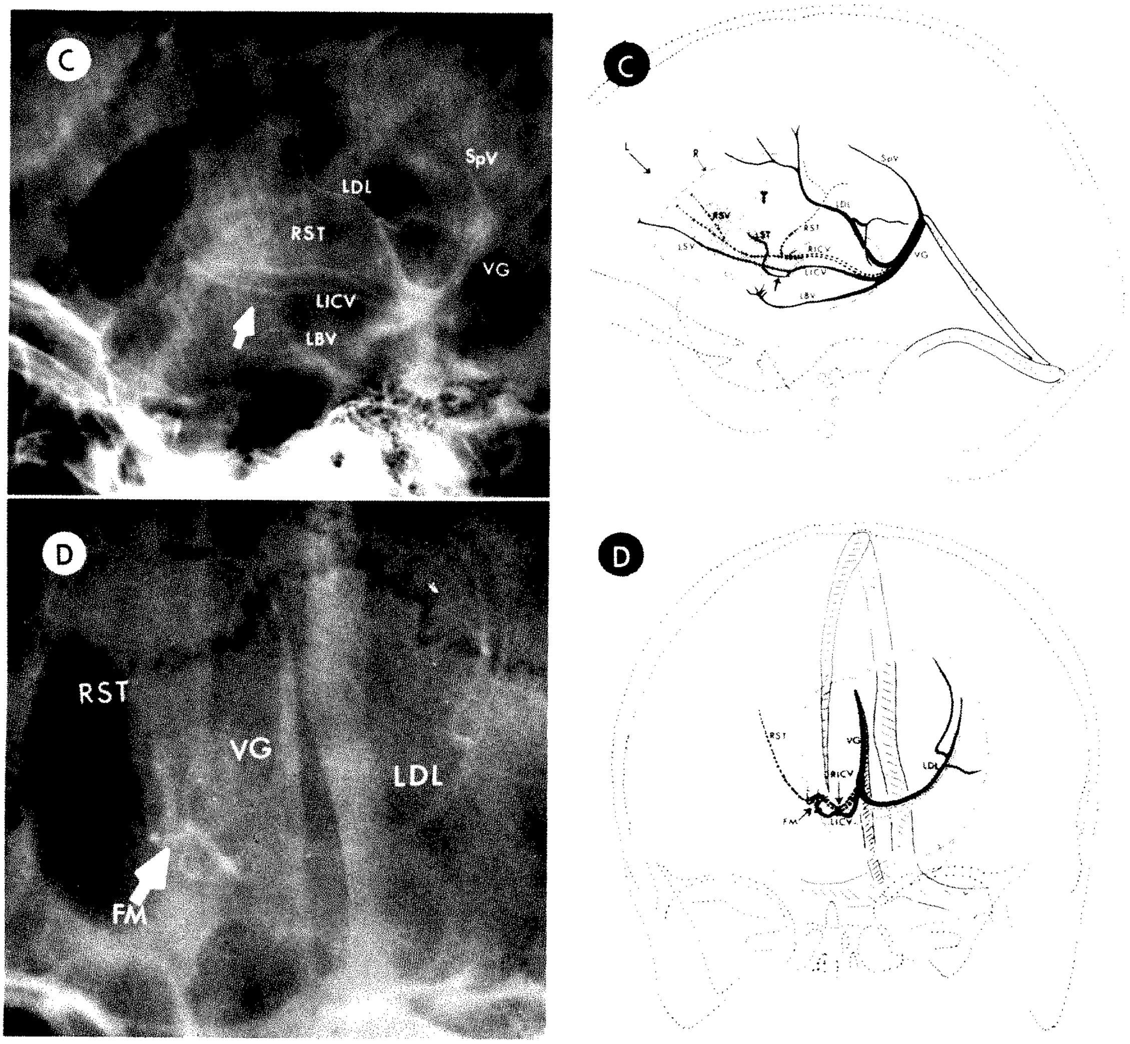

FIG. 4. (*C*) Venous phase—lateral view and line drawing of it. The roentgenogram is one of several venous phases. Information from this and the other films are included in the diagram. A large direct lateral subependymal vein on the left side (LDL) simulates an elevated internal cerebral vein. The left internal cerebral vein (LICV) is markedly depressed and flattened. The elongated left direct lateral vein and left septal vein (LSV) indicate marked enlargement of the left lateral ventricle. The tributaries of the left striothalamic stem (LST) are not evident. The stem itself is irregular and markedly depressed (arrow) at its junction with the internal cerebral vein. The junction, *i.e.*, the posterior border of the foramen of Monro, is displaced not only downward but backward. The splenial vein (SpV) is displaced upward. The left lateral basal vein (LBV) is normally situated. The subependymal veins of the right side are indicated in the diagram in dotted fashion. Elongation of the right septal vein (RSV), right striothalamic vein (RST) and depression of the right internal cerebral vein (RICV) indicate dilatation of the right lateral ventricle but to a lesser degree than the left. The great vein of Galen (VG) is also labeled. The ventricular portion of the tumor (T) is illustrated as shown on encephalography.

(*D*) Venous phase—anteroposterior view and line drawing of it. The roentgenogram shows rather marked rotation. Visualization of some of the subependymal veins is not entirely satisfactory and these have been illustrated to be consistent with the lateral projection. The elongated left direct lateral vein (LDL) is markedly displaced laterally and shows a wide sweep. Both internal cerebral veins (LICV, RICV) are displaced downward. The anterior end of the internal cerebral vein indicating the location of the foramen of Monro (FM) is markedly displaced toward the right. The right striothalamic vein (RST) is displaced toward the right. The distance between this vein and the mid-line great vein of Galen (VG) is about the same as the distance between the left direct lateral vein and the vein of Galen. However, the distance between the right striothalamic vein and the displaced foramen of Monro is much less than the distance between the foramen and the left direct lateral vein. The left ventricle must therefore be considerably larger than the right.

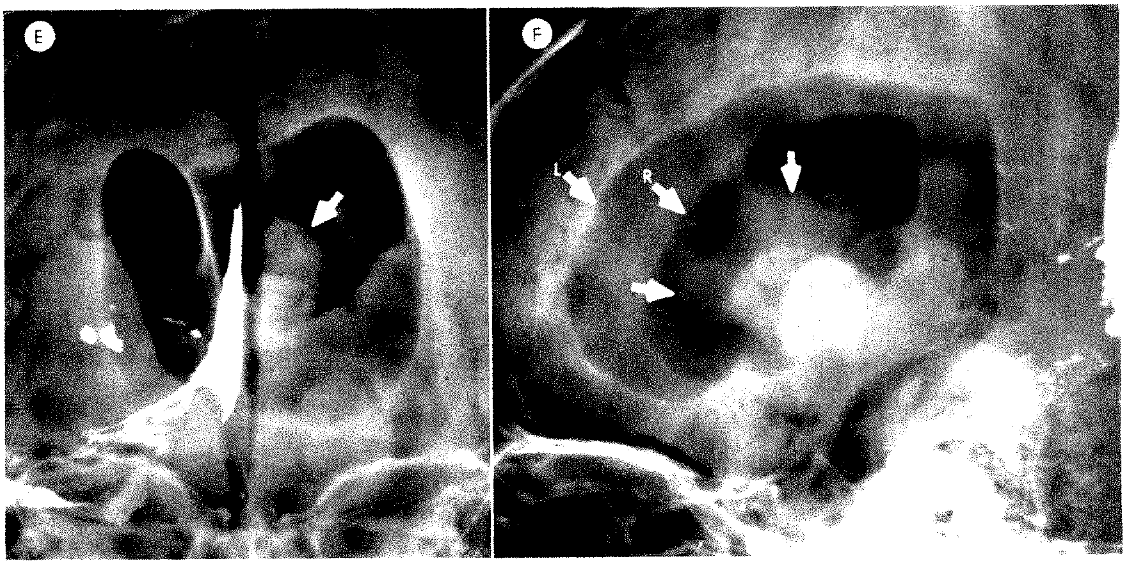

FIG. 4. (*E*) Pneumoventriculogram after unsuccessful attempt at pantopaque ventriculography—brow-up anteroposterior view. Subdural pantopaque is present above the tentorium and along the falx. An irregular tumor mass (arrow) extends upward into the markedly dilated left ventricle from the region of the foramen of Monro. The septum pellucidum is displaced to the right. The slight bowing of the septum pellucidum toward the left side is probably artefactual since the ventricles did not communicate and each was injected with air separately.

(*F*) Pneumoventriculogram—brow-up lateral view. The irregular tumor mass (unlabeled arrows) in the region of the foramen of Monro is evident. The left lateral ventricle (arrow L) is considerably larger than the right (arrow R).

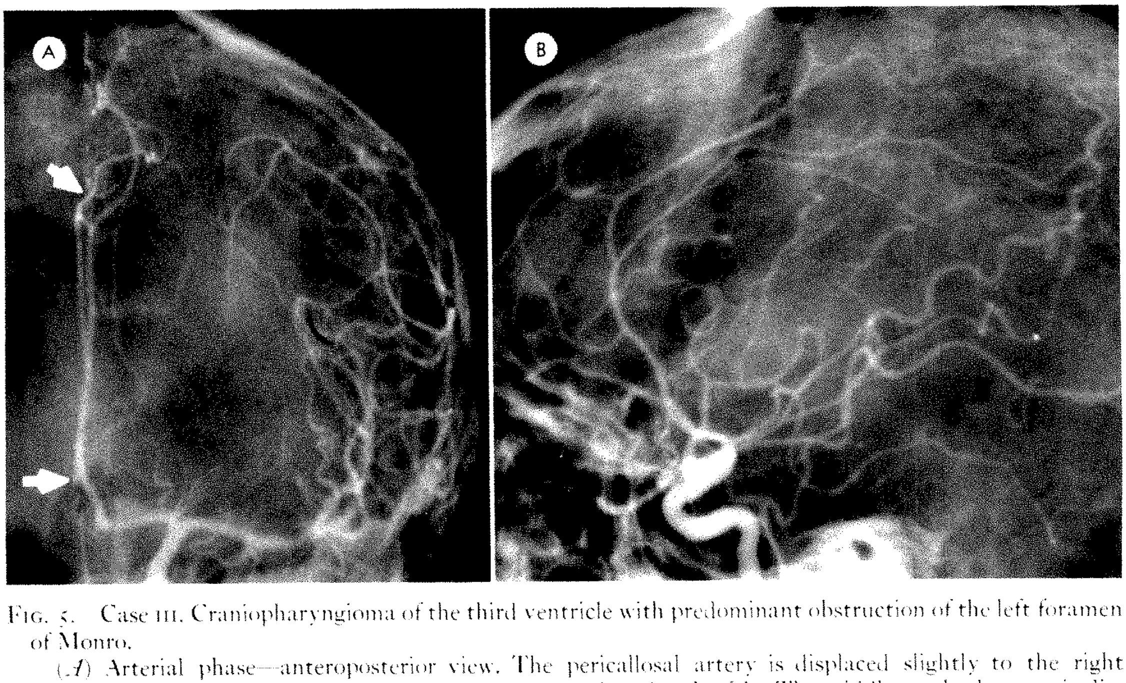

FIG. 5. Case III. Craniopharyngioma of the third ventricle with predominant obstruction of the left foramen of Monro.

(*A*) Arterial phase—anteroposterior view. The pericallosal artery is displaced slightly to the right (lower arrow) and returns to the mid-line (upper arrow) under the falx. The middle cerebral artery is displaced slightly laterally.

(*B*) Arterial phase—lateral view. The pericallosal artery shows a large wide, sweeping course. The sylvian vessels are within normal limits.

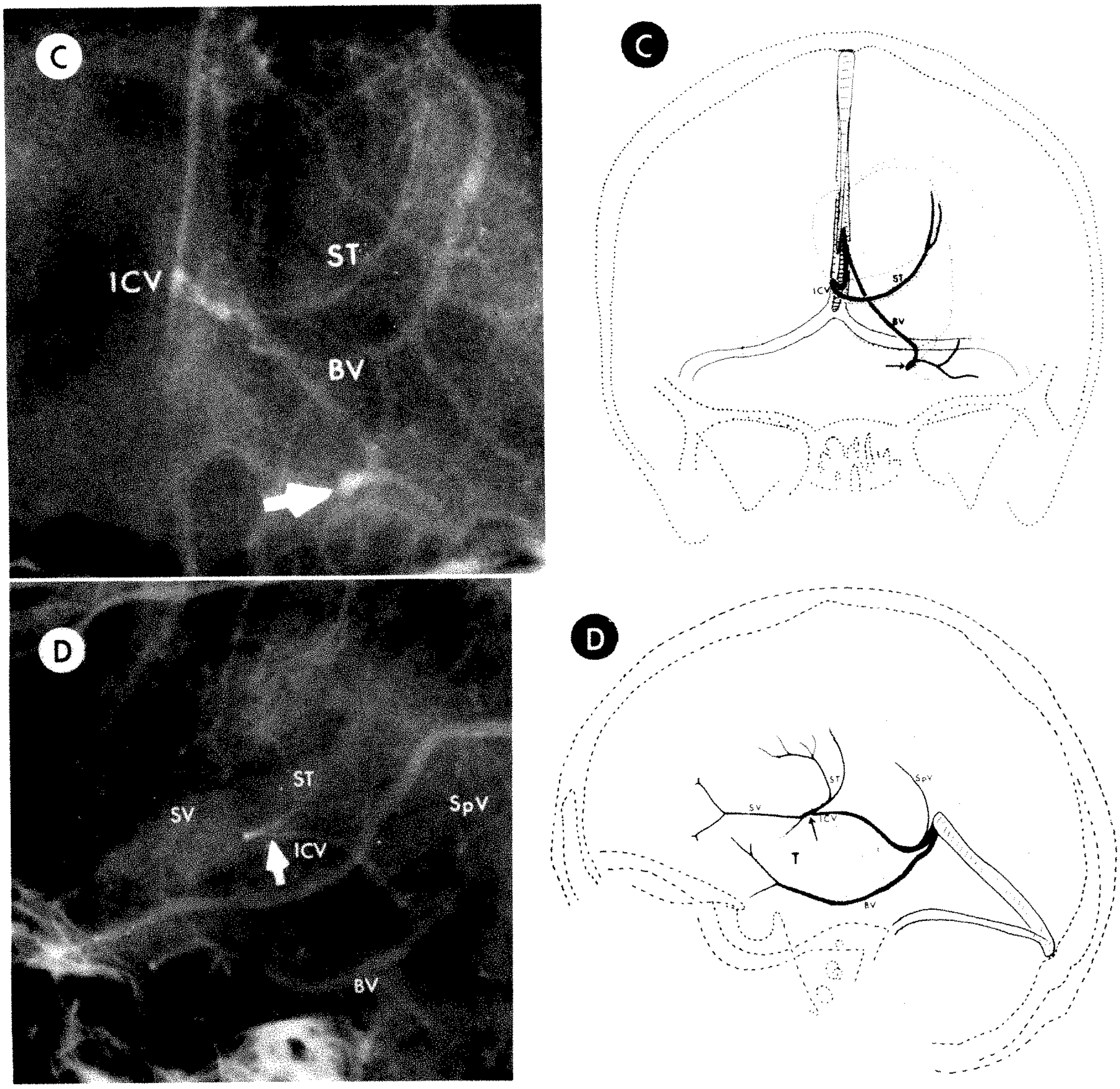

Fig. 5. (*C*) Venous phase—anteroposterior view and line drawing of it. The anterior end of the internal cerebral vein (ICV) is displaced toward the right side to a greater degree than the pericallosal artery. The striothalamic vein (ST) is displaced laterally and shows a wide sweeping course as it runs downward and medially. The junction of this vessel with the anterior end of the internal cerebral vein (ICV) indicates the location of the displaced foramen of Monro and the adjacent septum pellucidum. The anterior portion of the basal vein of Rosenthal (BV) is displaced laterally (arrow). The markedly dilated left lateral ventricle with displaced septum pellucidum is shaded.

(*D*) Venous phase—lateral view and line drawing of it. The septal vein (SV) and striothalamic vein (ST) are elongated. The anterior portion of the internal cerebral vein (ICV) is flattened and its anterior end (arrow) elevated. In this projection, the junction of the striothalamic vein and the internal cerebral vein forms an acute angle. This acute angle results from elevation of the roof of the third ventricle and depression of the floor of the lateral ventricle. The basal vein of Rosenthal (BV) is depressed slightly downward. The distance between the internal cerebral vein and the vein running around the splenium (SpV) is increased. The tumor (T) within the third ventricle is shaded.

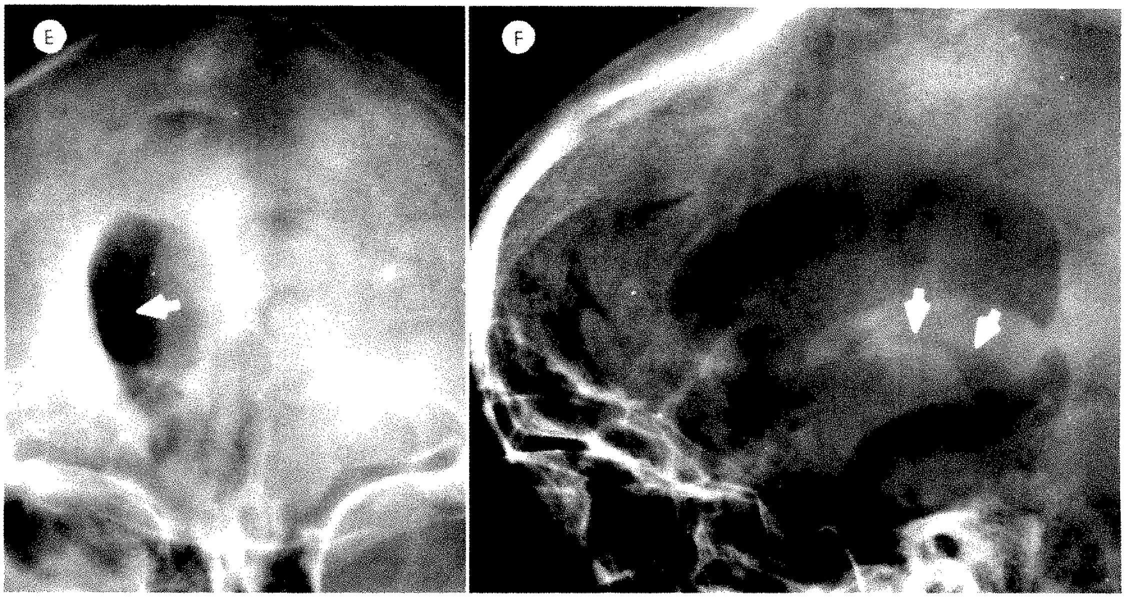

FIG. 5. (*E*) Pneumoencephalogram—brow-up anteroposterior view. No air entered the left lateral ventricle. The right lateral ventricle is moderately dilated with marked bowing of the septum pellucidum (arrow) into it from the opposite side.

(*F*) Pneumoencephalogram—brow-up lateral view. The right lateral ventricle is moderately dilated. A soft tissue mass occupies the anterior portion of the third ventricle. A cleft-like collection of air (arrows) is seen above and posterior to the tumor. The posterior clinoids and dorsum sellae are destroyed.

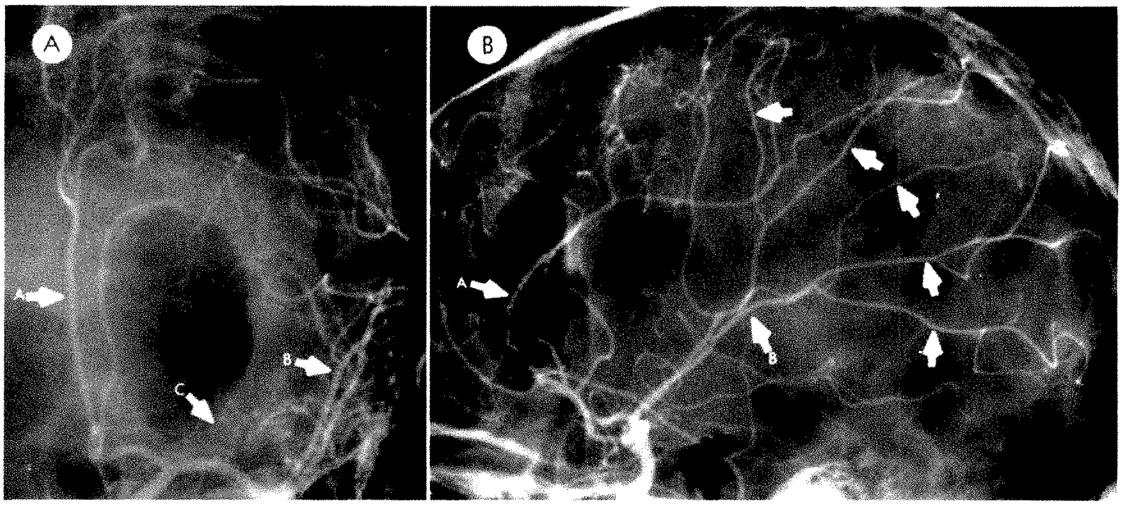

FIG. 6. Case IV. Huge cyst attached to the medial wall of the atrium of the left ventricle.

(*A*) Arterial phase—anteroposterior view. There is residual air in both frontal horns from a previous pneumoencephalography, the findings of which were not available at the time of angiography. The pericallosal artery and its branches (arrow A) are displaced to the opposite side, that is, to the right. The sylvian vessels (arrow B) are markedly displaced laterally. The striate arteries (arrow C) are flattened and tilted laterally.

(*B*) Arterial phase—lateral view. The pericallosal artery around the genu (arrow A) is bowed but not markedly elevated. Its course corresponds to the frontal horn of the smaller right ventricle. The posterior frontal and parietal branches of this artery are stretched and straightened. The insular portions of the sylvian vessels, particularly at the posterior insular or sylvian point (arrow B), are displaced anteriorly. The parietal branches of the middle cerebral artery are markedly stretched and spread apart (unlabeled arrows) simulating a large infiltrating mass.

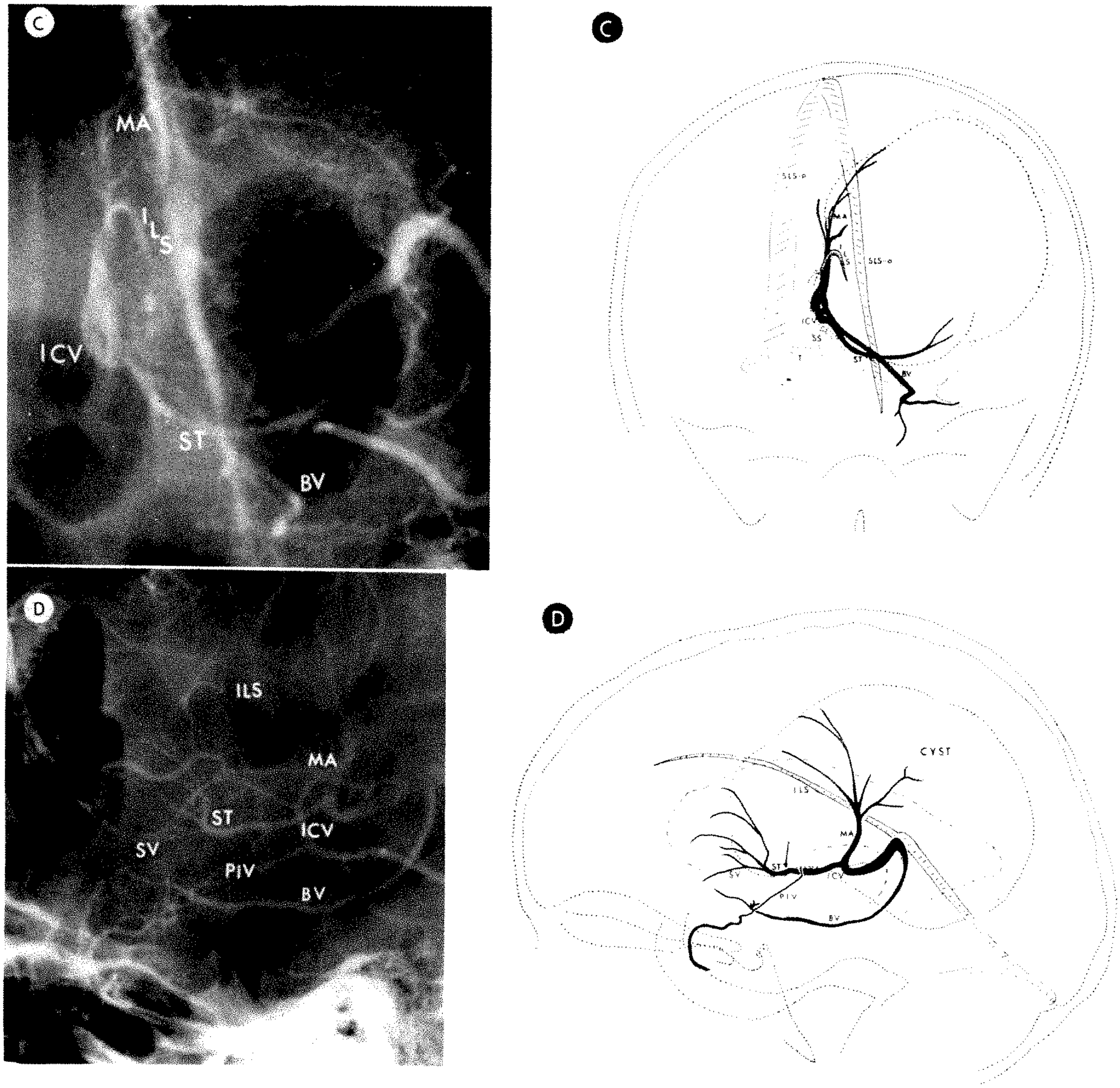

Fig. 6. (*C*) Venous phase—anteroposterior view and line drawing of it. At first sight, this appears to be a markedly rotated roentgenogram. Most of this is apparent, however, and is due to the fact that the posterior portion of the superior longitudinal sinus (SLS-p) is located unusually far to the right side. This is also true of the torcula (T) which shows an abnormal confluence of veins. The left side of the skull is considerably larger than the right. The striothalamic vein (ST) is markedly elongated and depressed. The internal cerebral vein (ICV) is displaced toward the right side. The medial atrial vein (MA) and its tributaries are elongated, elevated and also displaced toward the right. Other labeled structures are the anterior portion of the superior longitudinal sinus (SLS-a), the inferior longitudinal sinus (ILS), the straight sinus (SS) and the basal vein of Rosenthal (BV). The left lateral ventricle is shaded.

(*D*) Venous phase—lateral view and line drawing of it. The stem of the striothalamic vein (ST) is displaced downward. The medial atrial vein (MA) is formed by numerous tributaries which begin in the roof of the markedly dilated lateral ventricle. This vein joins the mid-portion of the internal cerebral vein (ICV) at a right angle and produces a kink at this site. The internal cerebral vein is flattened and its anterior portion displaced markedly downward. The posterior insular vein (PIV) in the posterior limiting sulcus of the insula is displaced forward. Also labeled are the inferior longitudinal sinus (ILS), a small septal vein (SV), and the basal vein (BV). The arrow points to the junction of the striothalamic stem and the internal cerebral vein. The cyst within the atrium is shaded.

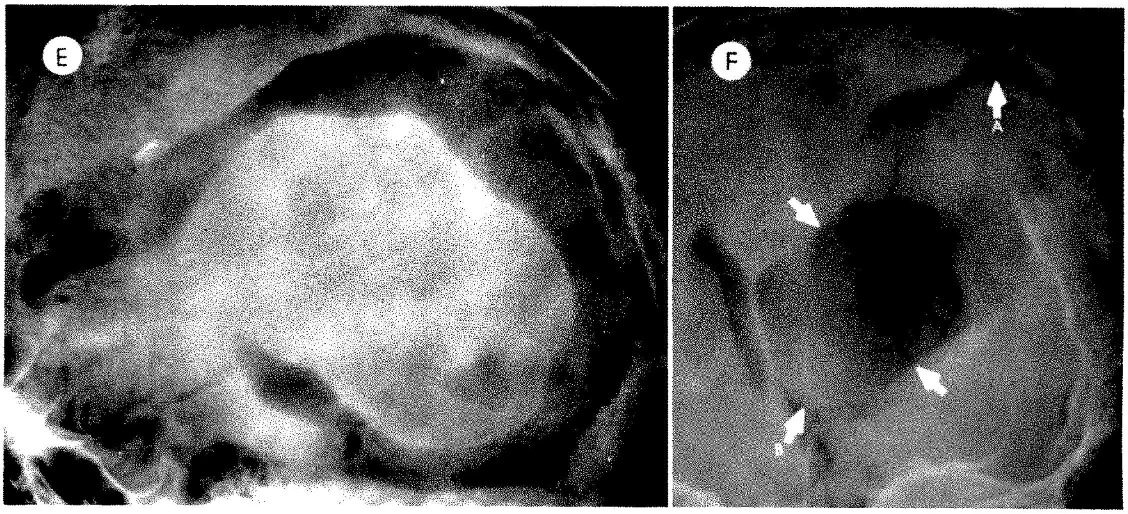

Fig. 6. (*E*) Pneumoencephalogram—lateral view. The atrium is hugely dilated and is occupied by a soft tissue mass. The tumor extends forward to the foramen of Monro.

(*F*) Pneumoencephalogram—anteroposterior brow-up view. The left lateral ventricle is markedly dilated even in its anterior portion (between unlabeled arrows). The septum pellucidum is deviated and inclined toward the right side. The air in the superior portion of the hugely dilated atrium outlines the upper margin of a spherical soft tissue mass (arrow A). Arrow B indicates the forward extension of the mass to the region of the foramen of Monro.

this patient showed extraordinary enlargement of the atrium as evidenced by the remarkable stretching and separation of the parieto-occipital arteries in association with marked medial displacement and elongation of the medial atrial veins. The frontal horn was enlarged but not to the same degree as the atrium. The marked depression of the anterior portion of the internal cerebral vein indicated that the third ventricle was not increased in size. While the huge size of the atrium suggested an expanding lesion within it, a mass protruding into the body of the ventricle anterior to the atrium can produce a similar appearance. It might be specifically noted that the anterior choroidal artery was not enlarged. This is in contrast to solid tumors in this area in which enlargement of this vessel is said to be characteristic.

Case v. This was the first admission of a 53 year old white male with chief complaints of numbness and weakness of the right lower extremity and mild headache of 3 weeks' duration. Neurologic examination showed bilateral early papilledema, a slow unsteady gait, a tremor of the right hand when outstretched, generalized weakness most marked in the distal portions of the extremities, impairment of vibratory and position sense in the right lower extremity and bilateral extensor response on plantar stimulation. A mild organic mental syndrome was present. Spinal tap showed a pressure of 430 mm. of water. The fluid was xanthochromic and contained 760 mg. per cent of protein. The red blood cell count on the spinal fluid was 5,000 cells per cubic mm. Carotid angiograms and pneumoencephalograms (Fig. 7, *A*, *B*, *C*, *D*, *E* and *F*) revealed evidence of a paraventricular mass adjacent to and extending into the body of the lateral ventricle on the left side. During the course of hospitalization, the patient developed a right homonymous hemianopsia. Radiation therapy was administered with some improvement and the patient was discharged without surgical intervention.

Comment. Carotid angiography in this patient showed disproportionate enlargement of the atrium with shift of the midline structures to the opposite side. These findings suggested obstruction posterior to

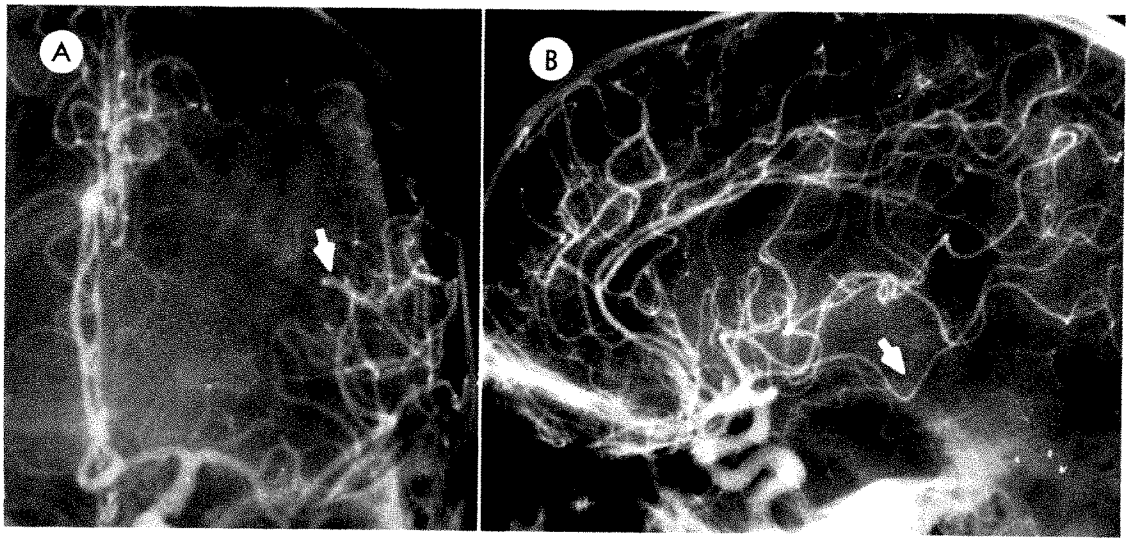

Fig. 7. Case v. Partial obstruction of the body of the lateral ventricle by a paraventricular neoplasm.

(*A*) Arterial phase—anteroposterior view. The pericallosal arteries are displaced toward the right. The arteries in the posterior portion of the sylvian fissure are displaced somewhat laterally (arrow).

(*B*) Arterial phase—lateral view. The course of the pericallosal arteries is within normal limits. The arteries in the posterior portion of the sylvian fissure are displaced forward. The intraventricular portion of the anterior choroidal artery in the anterior wall of the atrium (arrow) is displaced forward and downward.

the foramen of Monro. It was not possible to tell from the angiogram, however, the exact site and origin of the lesion. The changes in the direct lateral vein were probably the result of atrial dilatation although direct pressure from a mass lesion could not be completely excluded.

DISCUSSION

Dilatation of a lateral ventricle may occur as a result of diminution in the substance of one hemisphere, for example, in cerebral atrophy[4] and need not be on an obstructive basis. The clinical picture of these patients ordinarily does not suggest an expanding mass lesion and there is no evidence of increased intracranial pressure. From the roentgen point of view, significant displacement of the mid-line structures to the opposite side is unusual in such cases and the increase in size of the ventricle is rarely as marked as occurs in patients with obstructive lesions. The changes in the subependymal veins described above in obstructive unilateral hydrocephalus may also occur in this type of unilateral hydrocephalus. However, the course of the major arteries does not suggest ventricular dilatation under pressure. The anterior cerebral artery does not show a wide sweeping course and the midddle cerebral and posterior cerebral vessels are not elongated or stretched. A greater problem in making a specific diagnosis angiographically in patients with unilateral obstructive hydrocephalus is the recognition of the exact site of the tumor. The tumor may be quite small and not evident by any clear-cut arterial or venous deformity. It is often difficult with intraventricular and paraventricular lesions to be certain that the changes in the deep veins are not the result of direct pressure by the mass rather than simple dilatation of the ventricle and features of both may occur together. A confusing feature, particularly on angiography, of some cases of partial unilateral obstructive hydrocephalus is the fact that the ventricle distal to the obstruction, *i.e.*, between the site of obstruction and the foramen of Monro, may also show enlargement. This may occur with intraventricular lesions, *e.g.*, a large

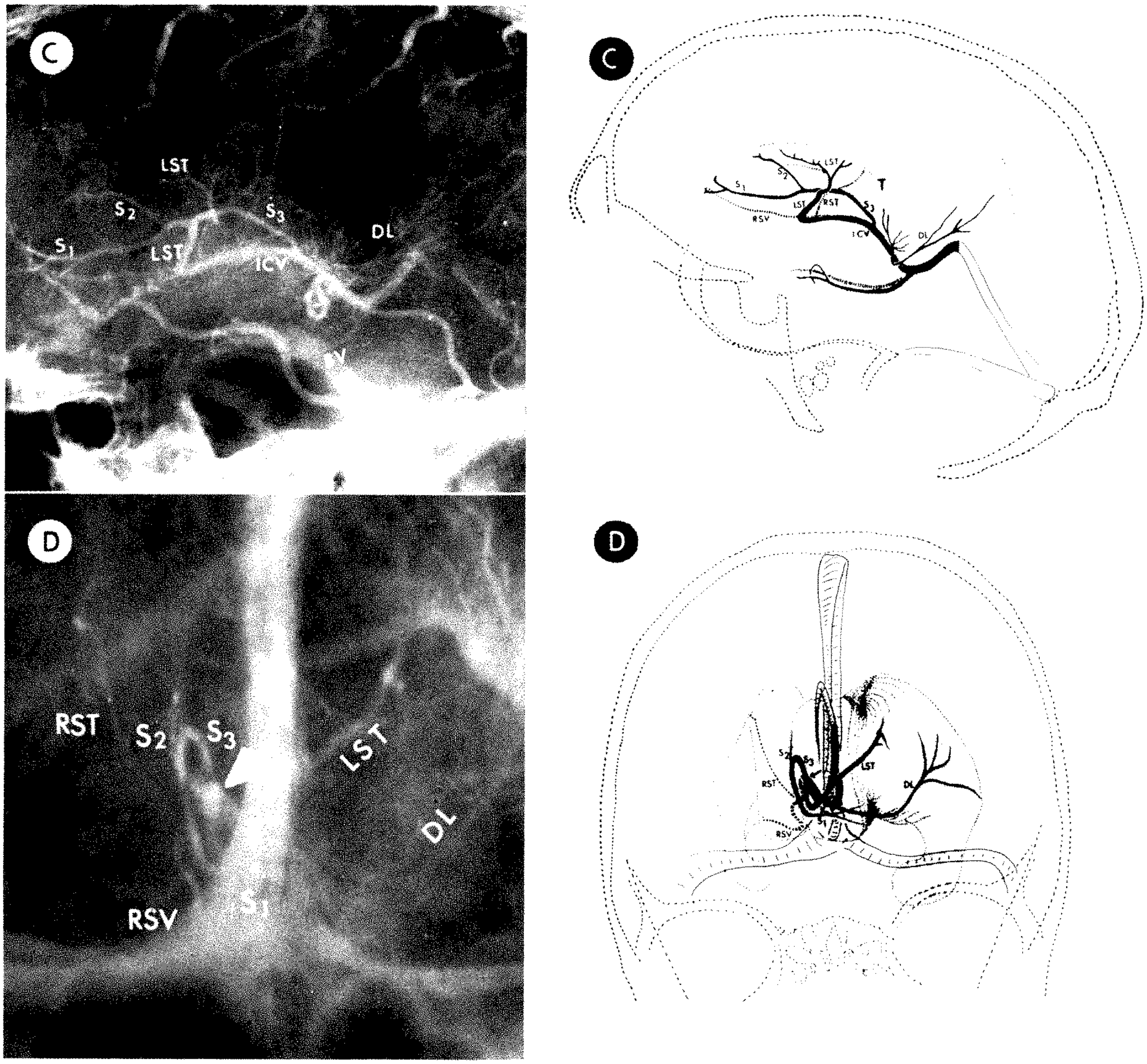

Fig. 7. (*C*) Venous phase—lateral view and line drawing of it. The septal tributaries (S_1 and S_2) unite to form the trunk of an anomalous septal vein (S_3) which drains into the mid-portion of the internal cerebral vein (ICV). The left striothalamic trunk (LST) and its tributaries cross the anomalous septal vein in this projection. Tributaries of the direct lateral vein (DL) arise in the posterior horn and lateral wall of the atrium and are markedly elongated. The septal (RSV) and striothalamic (RST) veins of the opposite or right side are also faintly seen. Both basal veins of Rosenthal (BV) can be recognized. The marked elongation of the direct lateral vein as compared with the striothalamic and septal veins is striking. Note that the internal cerebral vein is not flattened as would be expected with generalized dilatation of the lateral ventricle. The intraventricular protrusion of the tumor (T) is indicated as shown in the encephalogram.

(*D*) Venous phase—anteroposterior view and line drawing of it. The larger anterior tributaries of the anomalous septal vein begin in the anterior wall of the frontal horn (S_1) and run upward and backward in the displaced septum pellucidum after being joined by the small tributaries (S_2). The common trunk (S_3) runs downward to join the internal cerebral vein to the right of the mid-line (arrow). The stem of the left striothalamic vein (LST) is displaced medially, *i.e.*, toward the right, and does not show a wide sweeping course. The direct lateral vein (DL), however, is displaced laterally and downward and is obviously elongated. The disparity in the displacements between the direct lateral and striothalamic veins is again striking. The right septal vein (RSV) is also displaced toward the right. The right striothalamic vein (RST) is elongated in a vertical direction. The dilated left lateral ventricle is shaded.

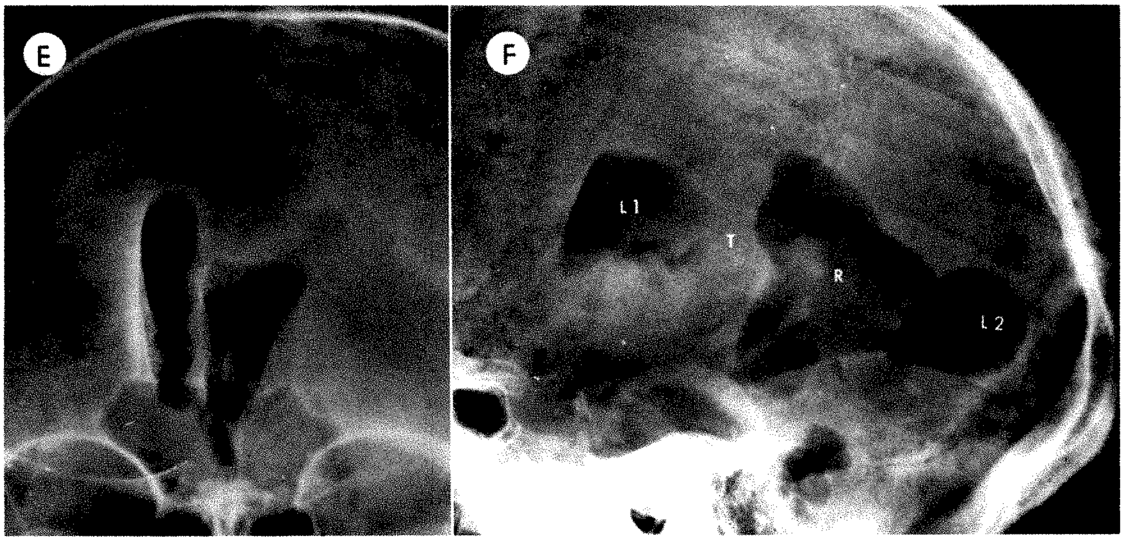

Fig. 7. (*E*) Pneumoencephalogram—brow-up anteroposterior view. The anterior portion of both lateral ventricles and the third ventricle are displaced toward the right. The left frontal horn is not markedly dilated. The roof of the left lateral ventricle is flattened and herniated under the falx. The right lateral ventricle is elongated in a vertical direction.

(*F*) Pneumoencephalogram—brow-down lateral view. There is a poorly demarcated soft tissue mass (T) which protrudes into the left ventricle between the body (L1) and the posterior horn (L2). The body of the lateral ventricle is somewhat dilated. However, the posterior horn is markedly and disproportionately dilated. In this projection, the dilated atrium (R) of the right lateral ventricle is projected over the unfilled portion of the left lateral ventricle.

meningioma of the atrium or with paraventricular tumors. The cause of this distal dilatation is often not clear and fortunately is usually considerably less marked than the dilatation that occurs at the site of or proximal to the lesion. In most instances of unilateral hydrocephalus, air studies, specifically ventriculography, will be required for exact delineation of the tumor mass. Unilateral obstructive hydrocephalus may also occur as a result of inflammatory changes in the lateral ventricle, *e.g.*, after a Torkildsen procedure. In such cases, the previous history and the absence of evidence of a mass on air studies are helpful in arriving at the correct diagnosis.

SUMMARY

A neoplasm may cause an increase in size of the cerebral hemisphere in two ways—as a result of the mass of the lesion *per se* or due to obstruction of one lateral ventricle. Distinction between these two processes is of importance for therapeutic reasons.

Unilateral obstructive hydrocephalus may be recognized on carotid angiography by findings indicating displacement of midline structures plus marked and disproportionate increase in the size of all or a portion of one lateral ventricle. The pericallosal artery, the internal cerebral vein, and the septal veins are displaced to the opposite side. The internal cerebral vein is flattened and the venous angle is widened. The individual subependymal veins are elongated and separated from each other with exaggerated curved courses concave toward the ventricle. In the anteroposterior projection, separation of these veins is shown by an increase in the transverse distance between veins of the medial and lateral group of subependymal vessels. Veins of the medial group are displaced medially while veins of the lateral group are displaced laterally. The corners of the ventricle are effaced as indicated by the wide sweeping course of the subependymal veins, particularly of the striothalamic, direct lateral and medial atrial veins.

The application of the basic principles underlying the diagnosis of unilateral obstructive hydrocephalus is illustrated by detailed analysis of selected cases.

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The authors would like to acknowledge the courtesy of Dr. Samuel D. Blum for permission to use the pneumoencephalogram of Case IV.

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EVALUATION OF ANGIOGRAPHY IN THE DIAGNOSIS OF SUPRASELLAR TUMORS*

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THIS study was instituted for the purpose of determining, insofar as possible, to what extent angiography may be of value in the diagnosis of tumors in the region of the sella turcica. The changes in the angiograms resulting from tumors in this area have been described in a number of publications,[5,6,7,9,11,12,13,17] the account by Chase and Taveras[3] being especially noteworthy. Relatively little has been written, however, concerning the actual incidence of such changes. It is this aspect of the subject that we propose to deal with in this presentation.

All authors agree that the blood vessels most often affected are the carotid siphon and the first part of the anterior cerebral artery. Changes involving the middle cerebral, posterior communicating and posterior cerebral and anterior choroidal arteries, and the internal cerebral and basilar veins occur less frequently. Of considerable importance in the interpretation of seemingly minor changes is the fact that many of the cerebral blood vessels, including both the intracranial and extracranial components of the internal carotid artery and the first part of the anterior cerebral artery, are subject to a wide range of normal variation. This has been recognized by many observers and is well illustrated in the article by Greitz and Lindgren.[8] To cope with this problem, various measurements were made by Udvarhelyi, Langfitt and Cox[16] in an attempt to establish objective criteria for evaluation of the position and configuration of the terminal carotid artery and the anterior cerebral artery. They concluded that only the angulation of the proximal anterior cerebral artery in the anteroposterior projection with respect to the horizontal plane, or its equivalent, the distance between the bifurcation of the internal carotid artery and the junction of the anterior cerebral and anterior communicating arteries in the vertical plane, was useful in this respect, and even this only to a limited degree. These authors also measured the distance between the bifurcation of the internal carotid artery and the midsagittal plane and found a mean value of 17.4 mm., with variations between 5 and 32 mm. in normal cases. These figures are germane to this presentation since we utilized this measurement in an analysis of our own material. Curtis[4] had earlier reported an average figure of 14 mm. with variations between 8 and 20 mm. In our own experience the average value was 15 mm. with variations between 8 and 18 mm.

In evaluating displacement of the cavernous segment of the internal carotid artery, the data compiled by Bull and Schunk[2] were used as a basis of comparison. We found it most feasible to determine the position of this vessel in the anteroposterior plane by measuring the distance between the midline and the medial border of the carotid artery at the level of the loop of the siphon. Normally, this figure varies between 5 and 11 mm., according to Bull and Schunk. Estimation of the position of the intracavernous portion of the artery in the lateral projection was much more difficult, especially in the group of pituitary adenomas in which the adjacent bony roentgenographic landmarks were often distorted and ill defined. Insofar as possible, the distance between the posterior surface of the dorsum sellae and the anterior border of the internal carotid artery at the level of the anterior clinoid process was utilized in assessing displacement of the vessel in this plane. Bull and Schunk found that this figure normally varied between 12 and 24 mm.

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MATERIAL

A total of 30 cases consisting of 20 pituitary tumors, 7 suprasellar meningiomas, 2 craniopharyngiomas and 1 chordoma was available for study.

PITUITARY TUMORS

Included in the group of pituitary tumors were 18 chromophobe adenomas, 2 of which exhibited evidence of hemorrhagic infarction (pituitary apoplexy), 1 eosinophilic adenoma without extrasellar extension and 1 malignant neoplasm. The nature of the lesion was verified histologically in 13 cases, 1 of which was operated upon as a recurrence in this clinic. In the remaining 7 cases, the clinical, roentgenographic and ophthalmologic features established the diagnosis beyond reasonable doubt. Extensions into the middle or anterior fossae occurred in 5 instances. The appearance of the sella turcica did not conform to the usual pattern of a pituitary tumor in 3 cases. Six patients were subjected to bilateral arteriography so that a total of 26 arteriograms was available for analysis; in 1 case only a lateral projection was used.

It is not the practice in this clinic to investigate all cases of pituitary tumor by means of arteriography as a matter of routine. A sizable portion of the material in this study exhibited features which differed in some way from the usual pattern of a pituitary adenoma so that further study, including arteriography and, in some instances, pneumoencephalography, appeared warranted. It should be apparent, therefore, that we are not dealing here with a wholly unselected group of cases.

Data concerning the cavernous segment of the carotid artery were available in 18 cases. Judging its position by the distance between the midline and the medial border of the vessel at the level of the loop of the siphon in the anteroposterior plane, lateral displacement was observed in 11 cases. Of the 6 cases in which bilateral arteriograms were made, lateral displacement was demonstrable on both sides in 4; in the other 2 cases the vessel was displaced only on one side. As already indicated, attempts to evaluate displacement of the cavernous segment of the artery in the anteroposterior plane in accordance with the criteria of Bull and Schunk were much less satisfactory. The appearance of the vessel, however, was thought to be abnormal in 4 cases. In these it was stretched and uncoiled to a degree distinctly beyond that which could be interpreted as a normal variant (Fig. 1, *A–G*). This appearance was observed bilaterally in 1 case; of the other 3, a large middle fossa extension was present in 1, in which the vessel was also markedly displaced laterally in the anteroposterior projection.

Abnormalities referable to the supraclinoid component of the carotid siphon visualized in the lateral view occurred in 9 cases; it was elevated in 7 (Fig. 2, *A–D*) and flattened in 2 (Fig. 3, *A* and *B*). The artery was elevated bilaterally in 1 case and on one side only in 2 others in which bilateral angiograms had been made; in 3 of the 7 cases the tumor had extended into the middle fossa. Of the 2 patients who exhibited flattening of the supraclinoid carotid siphon, the tumor had grown into the frontal fossa in 1. A normal configuration was observed in 11 patients, in 3 of whom bilateral arteriograms had been done.

Lateral displacement of the supraclinoid segment of the carotid artery was determined by measuring the distance between its medial border at the site of bifurcation and the midline in the anteroposterior projection. Accepting a figure of 20 mm. as the upper limit of normal, this distance was exceeded in 4 of the 18 cases in which this measurement was available. In 2 of these 4 cases, bilateral angiograms had been made; in each the vessel was displaced laterally only on one side.

Elevation of the anterior cerebral artery either at an angle or in a curvilinear manner with the concavity directed inferiorly was present in 11 of the 19 cases in which anteroposterior projections were available

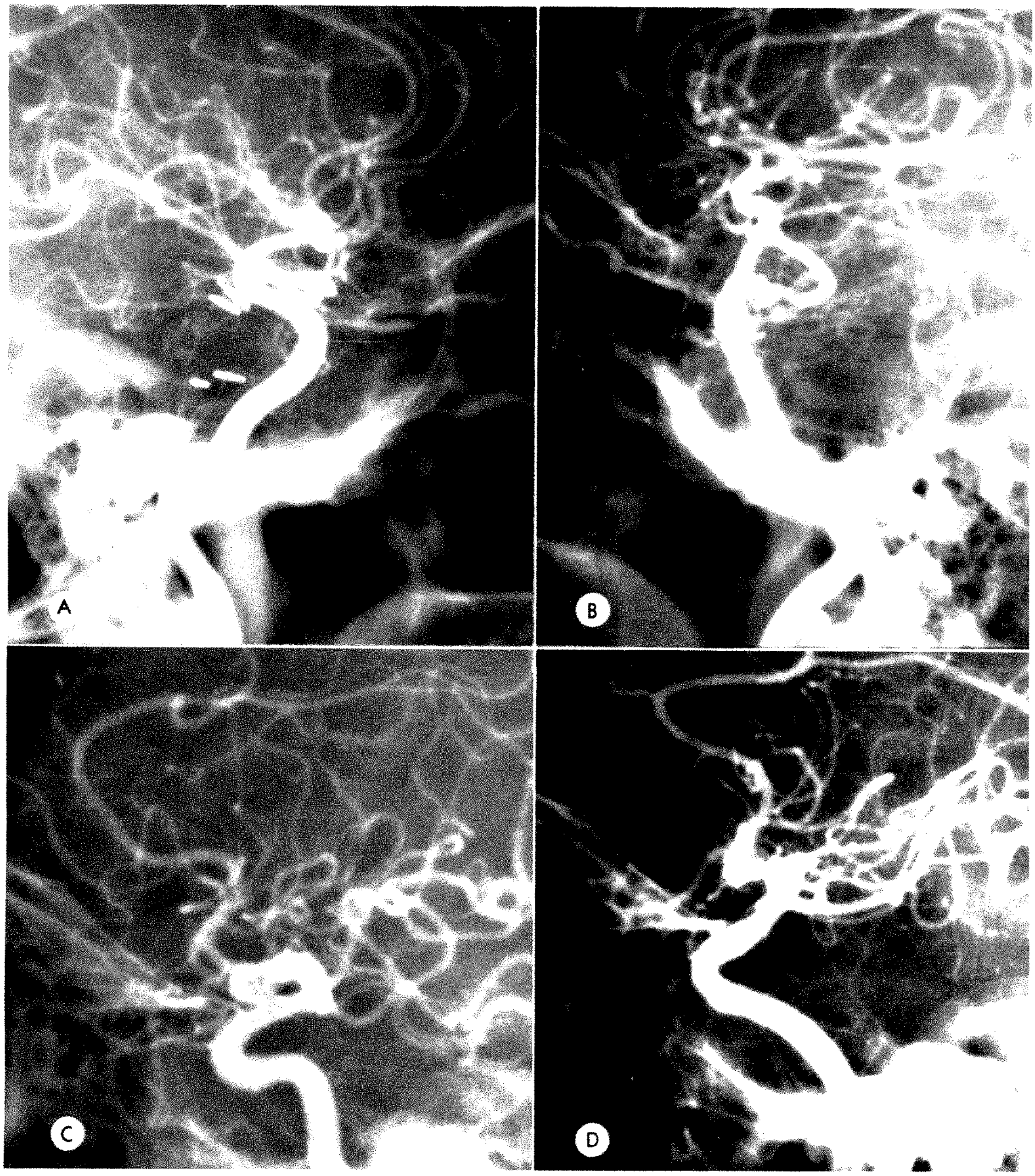

FIG. 1. Obliteration of normal contour of cavernous segment of internal carotid artery in cases of pituitary adenoma. (*A* and *C*) Normal side. (*B* and *D*) Abnormal side in 2 cases.

(Fig. 4, *A–F*). It was most pronounced in a patient with a huge midline bifrontal extension (Fig. 5). Of the 6 cases in which bilateral arteriograms were made, the vessel was elevated on both sides in 1, on one side only in 2 and on neither side in 3. In an additional case in which the cerebral circulation was visualized on both sides following a unilateral injection, both anterior cerebral arteries appeared in their normal position. Displacement of the pericallosal artery across the midline, to a marked degree, occurred in 1 instance in which the tumor extended into the frontal fossa (Fig. 6). The inferior portion of the pericallosal artery was shifted upward and backward in

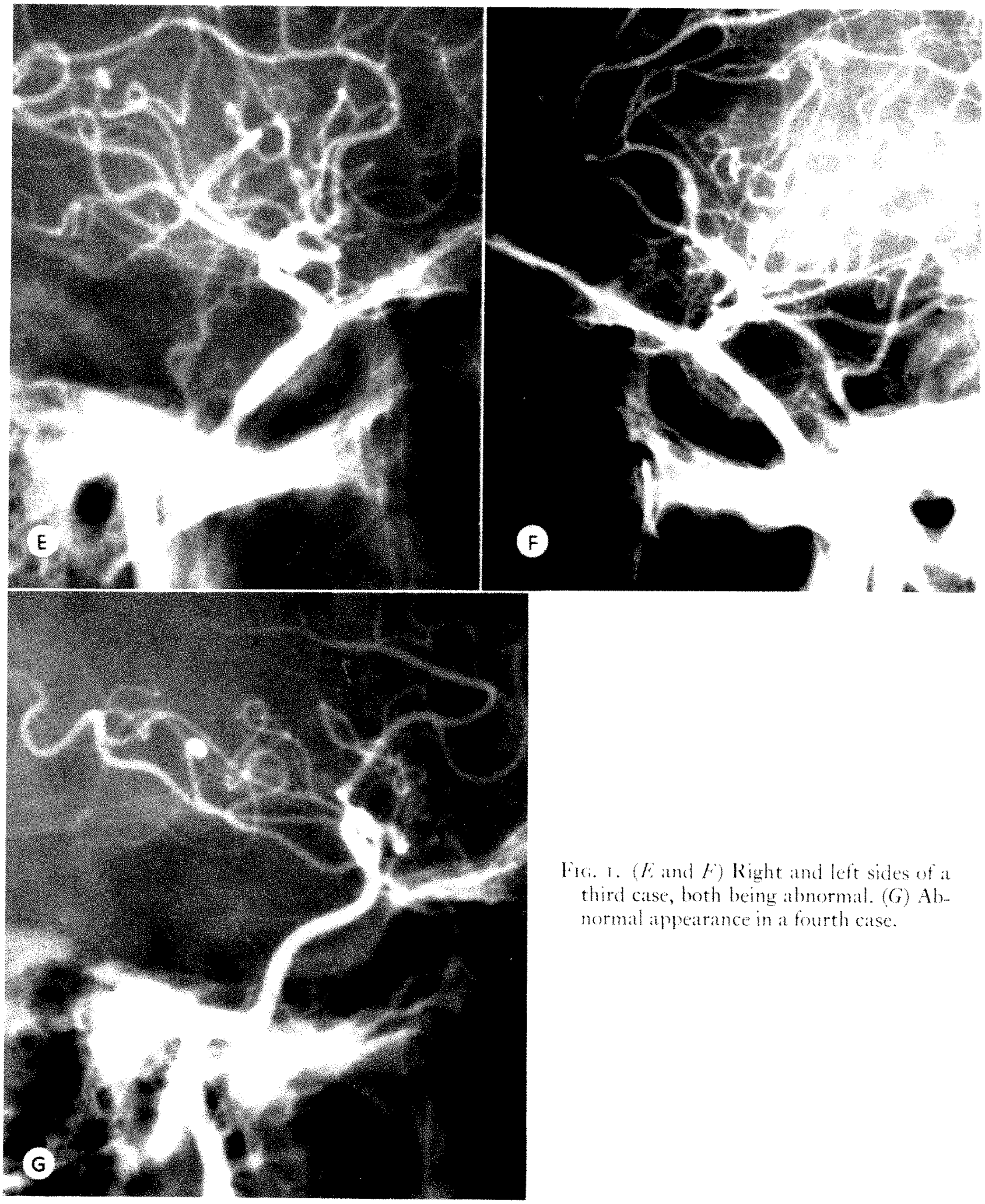

Fig. 1. (*E* and *F*) Right and left sides of a third case, both being abnormal. (*G*) Abnormal appearance in a fourth case.

this case and in another, also with a subfrontal extension (Fig. 7, *A* and *B*).

Allowing for the high take-off of the middle cerebral artery in cases in which the supraclinoid segment of the internal carotid artery was elevated (Fig. 8, *A* and *B*), abnormalities of the former vessel were apparent in 3 patients. It was elevated though flattened in 2 cases, in 1 of which the tumor had spread into the middle fossa (Fig. 9). Elevation of this vessel, discernible in both lateral and anteroposterior projections, occurred in another case with a middle fossa extension (Fig. 10, *A* and *B*).

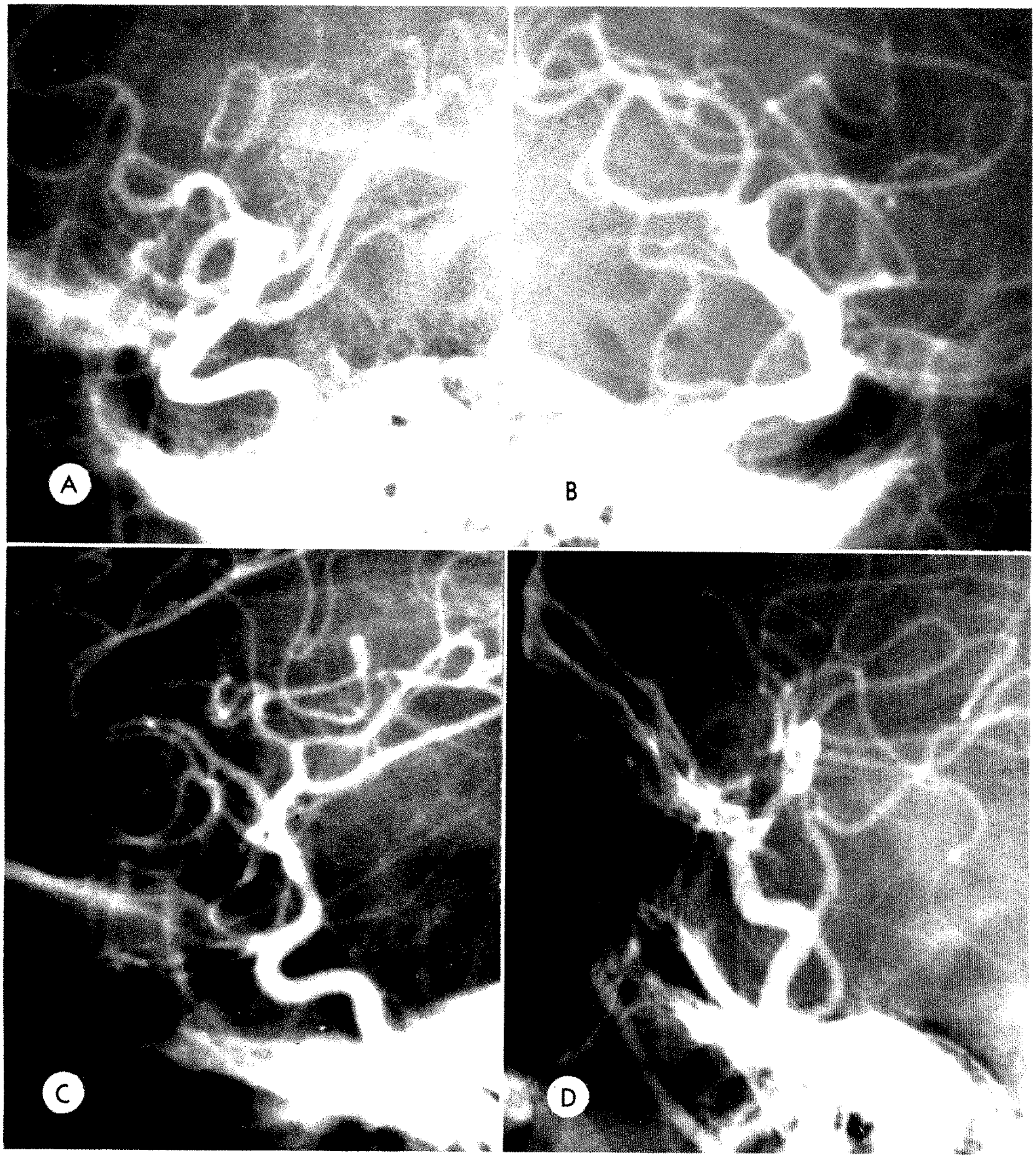

Fig. 2. (*A–D*) Elevation of supraclinoid component of internal carotid artery in association with pituitary adenoma. See also Figure 1, *B* and *D*.

The posterior cerebral artery was visualized infrequently in this series—in only 3 cases. It appeared abnormal only in 1 case, in which the tumor had grown into the middle fossa and elevated the artery (Fig. 11). No abnormalities of the vessel were noted in the anteroposterior projection.

The anterior choroidal artery was identified in 7 cases. It appeared definitely abnormal only in 1, in which there was a middle fossa extension and the artery appeared stretched and elevated (Fig. 10 *B*).

The internal cerebral vein was visualized in 16 cases and appeared normal in all but 2. In 1 of the latter, the tumor had extended into the middle fossa, and in the other, into

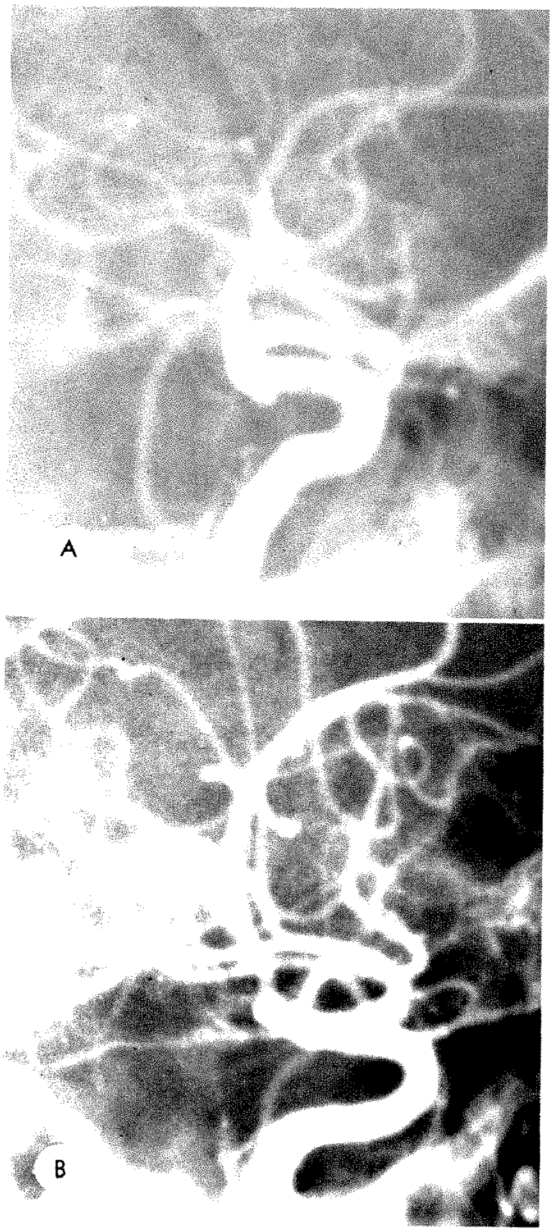

FIG. 3. (*A* and *B*) Pituitary adenoma with flattening of supraclinoid carotid siphon.

the frontal region. In each instance the vein was displaced superiorly and posteriorly as well as across the midline (Fig. 12, *A* and *B*). The basal vein was clearly identified only in 4 cases and appeared elevated in 2, in 1 of which the temporal fossa was invaded by tumor (Fig. 13).

Anatomic considerations suggested the possibility that an inverse relationship might be demonstrable between elevation of the supraclinoid segment of the carotid siphon and displacement superiorly of the anterior cerebral artery. While our data do tend to support this hypothesis, the material is not sufficient to be statistically significant. Thus the siphon was elevated in 4 of the 8 cases in which the anterior cerebral artery was normal in position (Fig. 8, *A* and *B*) and in 2 of the 11 cases in which the artery was displaced.

Tumor vessels were clearly visualized twice (Fig. 14, *A*, *B* and *C*). In each instance huge extrasellar extensions were present, in the temporal and frontal regions, respectively. In 1 the neoplasm was unequivocally malignant.

Pneumoencephalography was performed in 10 patients, revealing evidence of a tumor in all but 1. The exception was a patient with an eosinophilic adenoma whose vision was normal and who otherwise gave no indication whatsoever of an extrasellar extension.

SUPRASELLAR MENINGIOMAS

This group consisted of 7 cases, all of which were operated upon and the diagnosis verified histologically. The sella turcica was normal in each instance. In 2 patients hyperostoses were present in the region of the tuberculum sellae.

Bilateral arteriographies were performed in 3 cases, in 1 of which the opposite side was done after an interval of 2 years because of recurrent symptoms.

The cavernous portion of the siphon was displaced laterally in 2 patients. Its position in the anteroposterior plane was normal in all cases.

The supraclinoid segment of the siphon appeared elevated in 4 of the 7 cases. This abnormality was present only on one side in 1 of the 3 patients with bilateral angiograms and on neither side in the other 2. Lateral displacement occurred in none of the cases.

The first part of the anterior cerebral artery was displaced upward in the anteroposterior projection in all cases (Fig. 15, *A*–

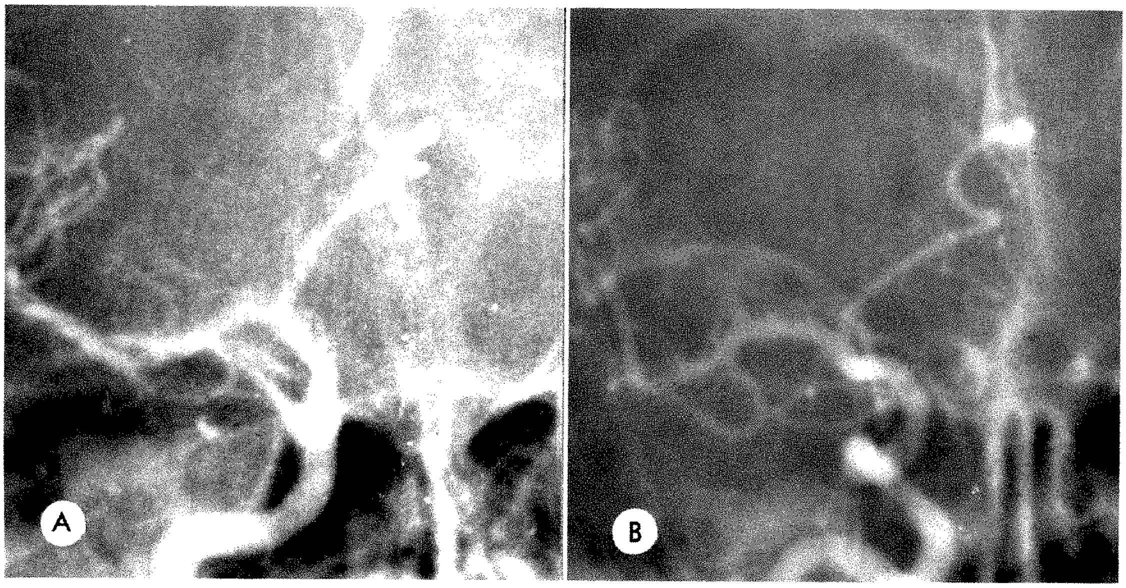

FIG. 4. (*A* and *B*) Elevation of anterior cerebral artery in cases of pituitary adenoma.

I). Elevation occurred only on one side in 1 of the patients in whom bilateral angiography was performed and on both sides in the other 2. It was also displaced upward and backward in the lateral projection in 1 case (Fig. 16).

The middle cerebral artery appeared normal in all instances as did also the anterior choroidal artery in the 3 cases in which it was identified; the posterior cerebral artery was visualized in none.

The venous phase was available for study in 5 of the 7 patients. The internal cerebral vein was visualized in all 5 and the basal vein in 3. No abnormality involving either vessel was noted.

Pneumoencephalography revealed the tumor in each of the 6 cases in which it was performed.

CRANIOPHARYNGIOMAS

The 2 cases of craniopharyngioma were both subjected to operation and the diagnosis substantiated histologically. Roentgenograms of the skull disclosed suprasellar calcification in one and erosion of the posterior clinoids and dorsum sellae in the other. The cavernous and supraclinoid component of the internal cerebral artery appeared normal in both instances. In neither case was the anterior cerebral artery elevated; it was visualized on both sides in 1 of the cases. In 1 patient the tumor was largely within the third ventricle resulting in obstructive hydrocephalus, reflected in the appearance of the pericallosal, middle cerebral and anterior choroidal vessels; in this case also, the pericallosal artery was displaced from the midline (Fig. 17, *A* and *B*). The posterior cerebral artery was not demonstrated in either patient. The veins were clearly visualized only in 1 case and appeared normal.

A ventriculogram in 1 case and a pneumoencephalogram in the other outlined the tumor.

CHORDOMA

The one case presented clinically and roentgenologically as a pituitary adenoma, the correct diagnosis being established at necropsy. The only definite angiographic abnormality was a lateral displacement of the intracavernous component of the carotid artery.

DISCUSSION

PITUITARY TUMORS

In only 3 of the 20 cases of pituitary adenoma were there no abnormalities recognized in the angiogram, 1 of the 3 being an

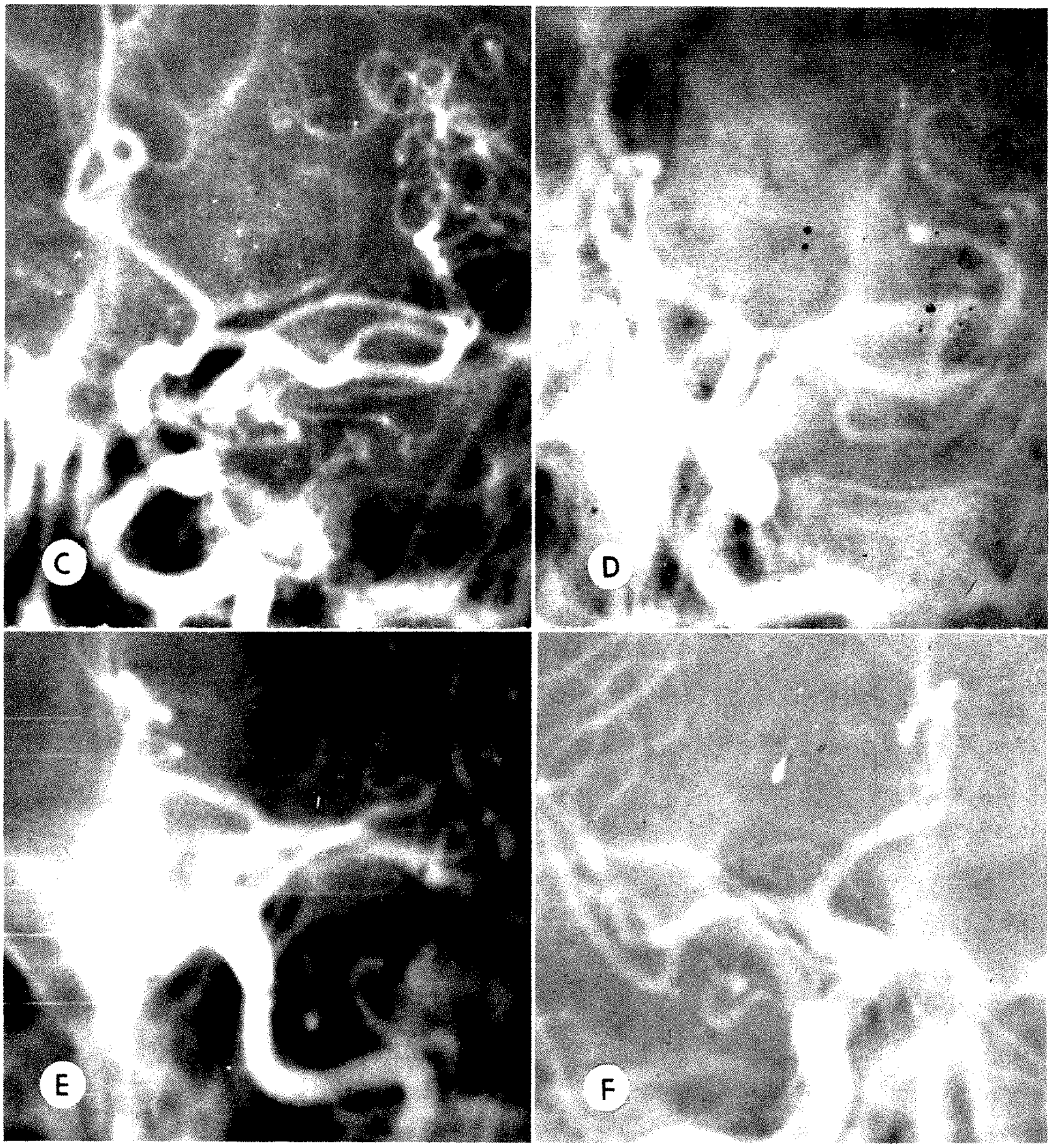

Fig. 4. (*C–F*) Elevation of anterior cerebral artery.

eosinophilic adenoma in which there was no evidence whatsoever of a suprasellar extension. This does not imply that the changes that were observed in 17 cases were sufficient to lead to an unequivocal diagnosis of a pituitary tumor in each instance. The most frequent changes caused by a pituitary tumor were lateral displacement of the cavernous segment of the internal carotid artery and elevation of the first part of the anterior cerebral artery, and, to a lesser degree, elevation and lateral displacement of the supraclinoid component of the internal carotid artery.

The significance of displacement of the cavernous portion of the internal carotid artery was studied by Bull and Schunk and the conclusion reached that such an abnormality is indicative of an extracerebral mass in the vicinity of the medial part of the middle cranial fossa. With specific reference to pituitary tumors, lateral displacement of the cavernous segment of the carotid artery was observed by these authors in 11 of 12 cases; in the remaining case the vessel was displaced medially. While lateral displacement of the cavernous portion of the carotid artery was also observed occasionally in other types of extracerebral tumor, its greatest incidence was in cases of pituitary adenoma. Chase and Taveras[3] concur in the value of this finding as being "fairly characteristic of pituitary adenoma as against other types of suprasellar tumor not originating within the sella turcica." It was also mentioned by List and Hodges[12] as a feature of tumors arising from within the sella turcica. Displacement in the anteroposterior plane was difficult to assess in our material. Bull and Schunk reported variable findings, anterior displacement in 5, posterior displacement in 2 and no displacement in 5 cases. It is noteworthy that the changes we observed affecting the cavernous segment of the carotid artery as well as other vessels were not necessarily symmetric. The occurrence of asymmetric vascular displacement in all types of sellar and suprasellar tumors has previously been noted by El-Banhawy and El-Nadi[7] and by Bakay and Bean.[1]

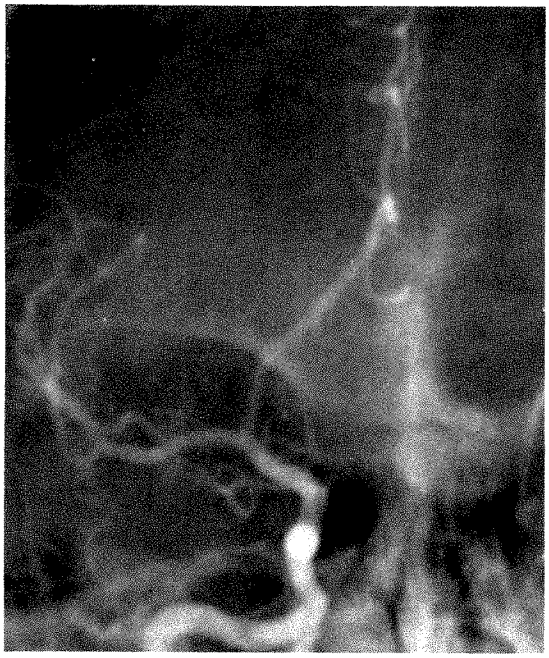

Fig. 5. Pituitary adenoma with large extrasellar extension showing marked displacement of anterior cerebral artery.

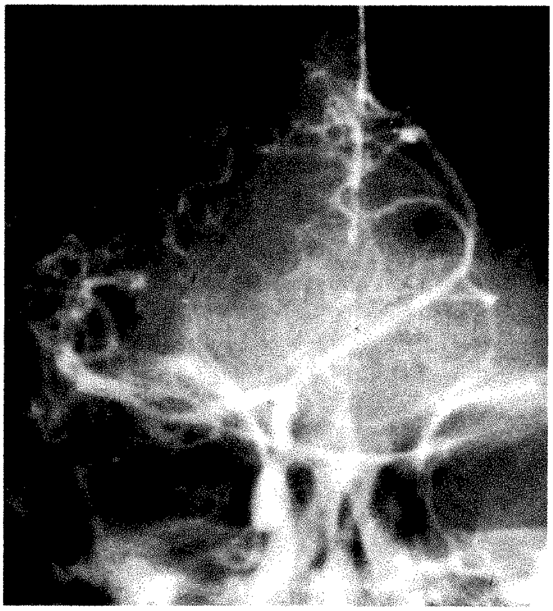

Fig. 6. Displacement of anterior cerebral artery across midline to a pronounced degree. Large frontal extension of a chromophobe adenoma. (By permission of *Neuroradiology Workshop*, Volume II, and Grune & Stratton, Inc., Publishers.)

Direction of growth rather than the type of tumor was suggested by Chase and Taveras[3] as a basis for a classification of the angiographic changes observed in suprasellar lesions. Thus, anterior suprasellar masses are characterized by elevation of the anterior cerebral arteries and posterior displacement of the supraclinoid segment of the internal carotid arteries, tumors extending directly upward by elevation and lateral displacement of the carotid siphon, elevation of the carotid bifurcation and, occasionally, lateral displacement of the anterior choroidal and/or posterior communicating arteries, and posterior suprasellar lesions, least amenable to recognition arteriographically, by lateral displacement and elevation of the anterior choroidal, posterior communicating and posterior cerebral arteries. Obviously, changes associated with growth in more than one direction may be observed. Abnormalities referable to the internal cerebral and basilar veins were infrequently observed by Chase and Taveras.[3] Specifically, elevation of the basilar vein occurred in 8 of their 89 cases of suprasellar tumor.

In attempting to evaluate the relative usefulness of pneumoencephalography and arteriography in the diagnosis of suprasellar masses, Bakay and Bean[1] studied 11 cases of chromophobe adenoma, in 2 of which the tumor was "confined to the sella with minimal growth above the diaphragma sellae." Widening of the anterior genu or "uncoiling" of the siphon was visible in the lateral projection in 10 of the 11 cases; elevation of the supraclinoid segment of the carotid artery occurred in half the cases and lateral displacement, "slight in many cases," in 9; upward displacement of the first part of the anterior cerebral artery on one or both sides was observed in 7 of the 9 cases with suprasellar extension; the posterior communicating artery was elevated and stretched in 1 case and the anterior choroidal artery and ganglionic branches displaced upward in 2. Pneumoencephalography demonstrated suprasellar extension in all cases, leading the authors to conclude that in cases of pituitary adenoma, "angiography supplements but does not supplant air studies."

Uncoiling and stretching of the infraclinoid part of the carotid siphon occurred in 4 of our cases. Obliteration of the normal contour of the siphon has been observed by others and is illustrated in the articles of Wickbom,[17] and Money and Vanderfield[13] and of Chase and Taveras.[3]

Reference has already been made to the study of Udvarhelyi, Langfitt and Cox,[16] in which measurements of several arbitrarily selected angles and distances were made in normal and pathologic cases to determine the validity of diagnosing suprasellar masses on the basis of displacement of the terminal internal carotid artery, its bifurcation and/or the proximal anterior cerebral artery. Only the degree of elevation of the anterior cerebral artery in the anteroposterior projection, it was concluded, was of differential diagnostic significance. The authors expressed doubt concerning the diagnostic value of variations in the course of the internal carotid artery and the position of its bifurcation. By their criteria the angiograms were unequivocally abnormal in only 7 of 22 cases of pituitary adenoma. In an additional 9 cases the angiographic study revealed a "doubtful" abnormality.

The conclusion reached on the basis of a

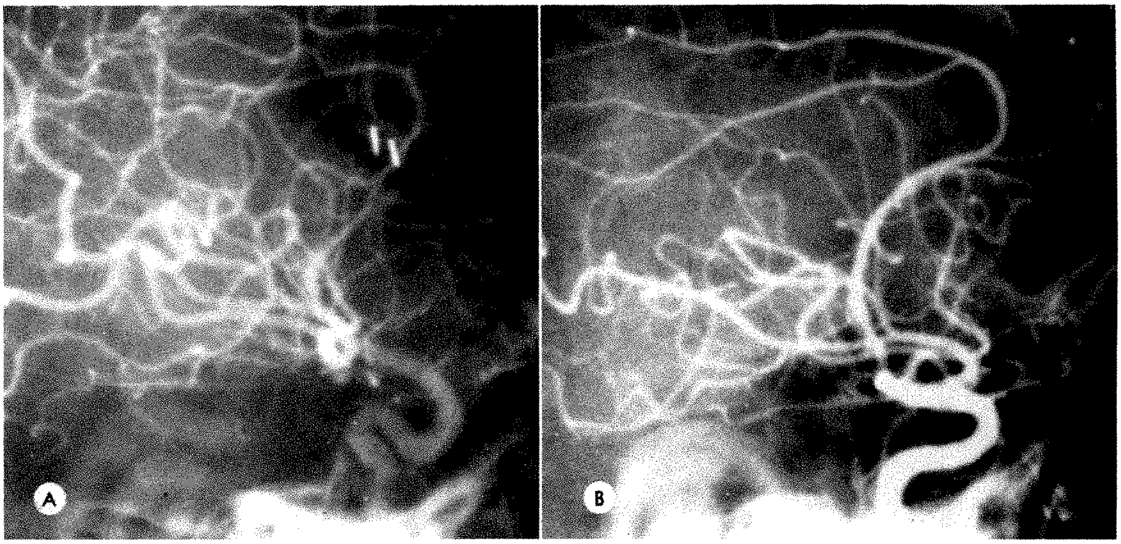

Fig. 7. (*A* and *B*) Pituitary tumors with large extrasellar components showing displacement of pericallosal artery upward and backward.

statistical analysis that apparent displacement of the supraclinoid segment of the internal carotid artery is without diagnostic significance is at variance with the opinions expressed by virtually all other authors. While we share the majority belief that changes in the terminal internal carotid artery can and do occur in suprasellar lesions, we are well aware of the difficulties that may arise in distinguishing between slight abnormalities of vascular configuration and normal variants. In fact, our data may be taken as evidence of this. Thus, we observed deviation of the supraclinoid segment of the internal carotid artery in only 7 of 20 cases, in 3 of which the tumor had extended into the middle fossa; similarly, lateral displacement occurred in only 4 of 18 cases. We are, therefore, inclined to pay heed to the note of caution sounded by Udvarhelyi *et al.*[16] Their figures for the distance between the bifurcation of the internal carotid artery and the midsagittal line (5–32 mm.) in normal cases show greater variation than those of Curtis[4] (8–20 mm.) and our own (8–18 mm.).

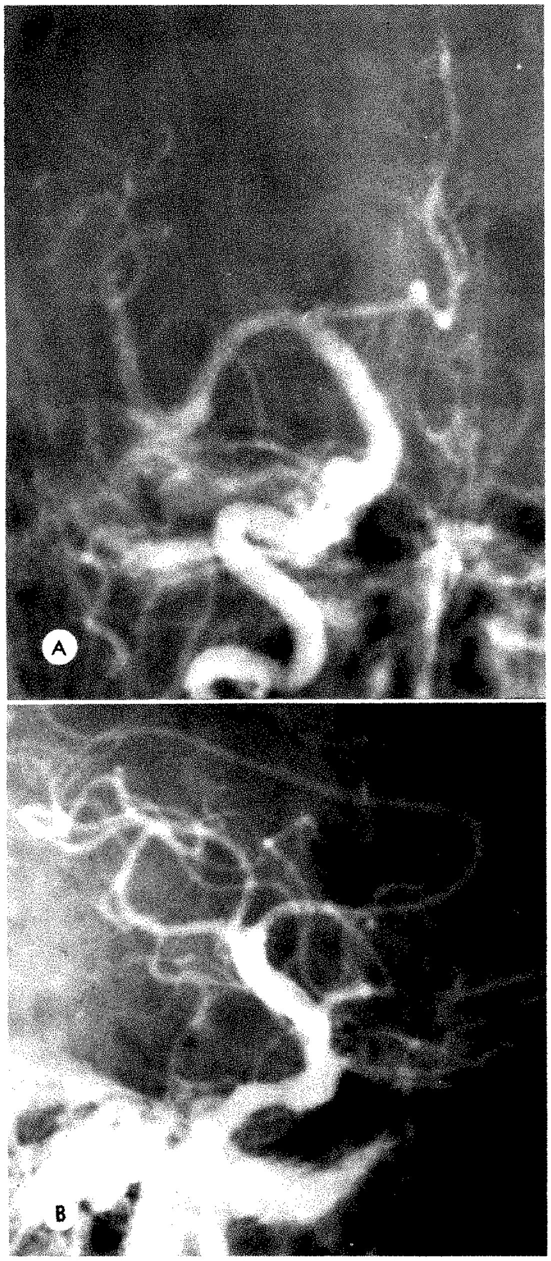

Fig. 8. (*A*) Anteroposterior and (*B*) lateral projections in a case of pituitary adenoma with high take-off of middle cerebral artery. The siphon is elevated and the anterior cerebral artery is norma in position.

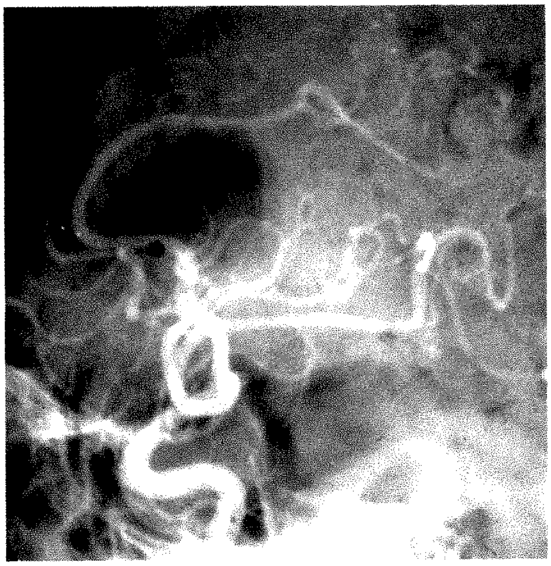

Fig. 9. Flattening of middle cerebral artery. Pituitary adenoma. See also Figure 1*B*.

The demonstration of a tumor stain in 2 cases is most unusual. To our knowledge this has been reported previously in only 1 other case.[10] Both of our patients had large extrasellar extensions and in 1 the lesion was malignant.

Judging from our experience and from pertinent reports in the literature, angiography plays an important but not necessarily decisive role in the diagnosis of pitui-

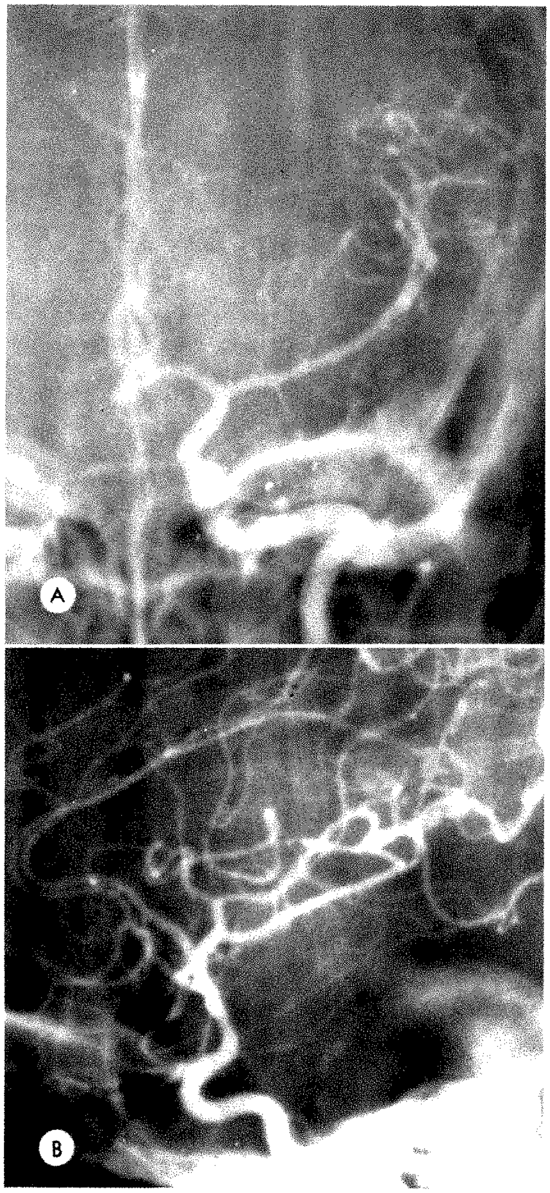

FIG. 10. (*A* and *B*) Chromophobe adenoma with middle fossa extension showing elevation of middle cerebral artery in both projections. In the lateral projection the anterior choroidal artery appears stretched.

tary tumors. Obviously, the size of the lesion and its direction of growth are of paramount importance. Unequivocal abnormalities may be recognized in about one-third to one-half of the cases. Lateral displacement of the cavernous segment of the internal carotid artery and elevation of the first part of the anterior cerebral artery are probably the most common and significant changes. Abnormalities involving the supraclinoid component of the internal carotid artery do occur, but are often difficult to evaluate. Bilateral angiography may be expected to yield a higher incidence of abnormality since asymmetric changes are not uncommon. Fortunately, in the vast majority of cases, the clinical features, including visual and endocrinologic manifestations and plain roentgenograms of the skull establish the diagnosis so that angiography is unnecessary. It is indicated in all cases in which an aneurysm enters into the differential diagnosis. It is also extremely informative in cases of massive extrasellar extension. In those instances of suspected suprasellar tumor in which angiography is not definitive, pneumoencephalography should be performed.

SUPRASELLAR MENINGIOMAS

Upward displacement of the horizontal segment of the anterior cerebral artery and,

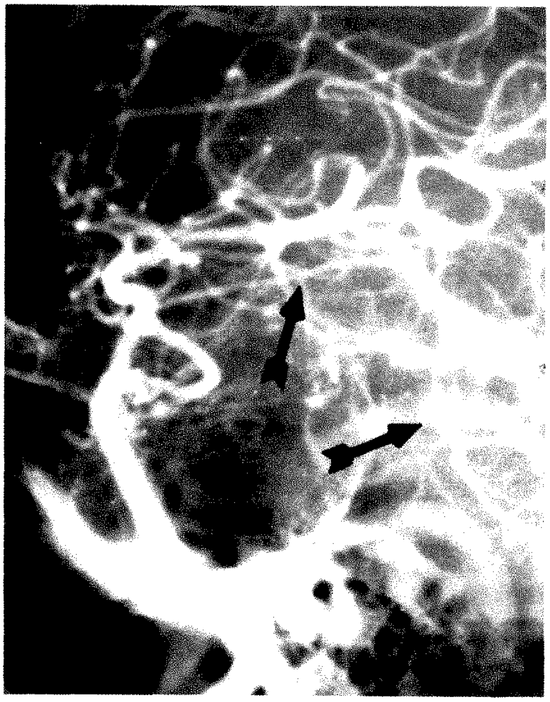

FIG. 11. Elevation of posterior cerebral artery (arrows) in a pituitary adenoma with extension into temporal fossa.

less frequently, elevation of the supraclinoid component of the internal carotid artery were the characteristic features observed in this series. At times it was difficult to be certain whether the degree of elevation of the anterior cerebral artery was within or beyond the limits of normal. It has been suggested by Chase and Taveras[3] that the anterior cerebral artery just distal to the siphon in the lateral projection loses its normal downward convexity in the presence of an anterior suprasellar tumor. We are inclined to doubt the validity of this observation since, in our experience, this vessel normally exhibits a good deal of variation and, may, in fact, be flat or slightly concave anteriorly. Such variation is depicted in the articles of Curtis,[4] Greitz and Lindgren[8] and of Tönnis, Brandt and Walter.[14]

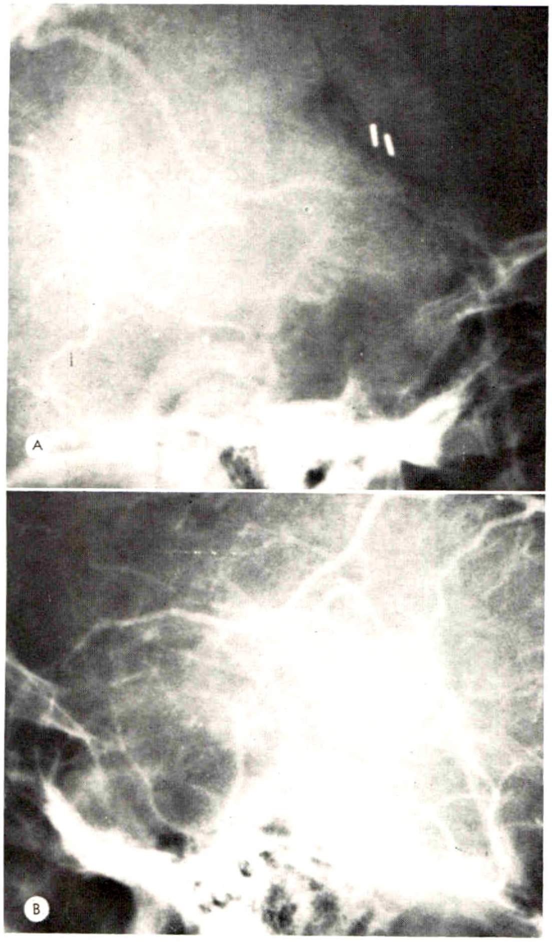

Fig. 12. (*A* and *B*) Displacement of internal cerebral vein in cases of pituitary adenoma.

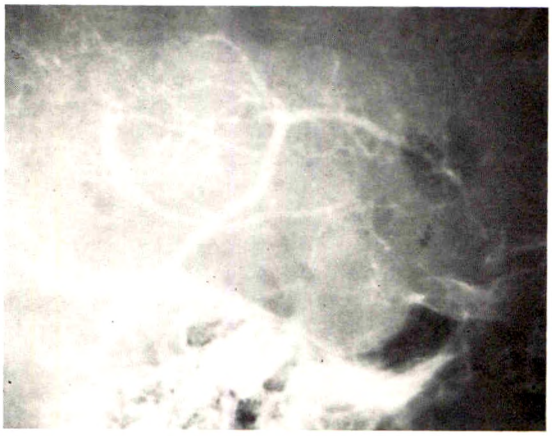

Fig. 13. Case of pituitary adenoma with elevation of basal vein.

Lateral displacement of the cavernous part of the internal carotid artery occurred in 2 of our 7 cases. Bull and Schunk[2] observed it only once in 8 cases.

Although anterior suprasellar tumors are said to displace the supraclinoid segment of the internal carotid artery posteriorly and inferiorly, this was not apparent in any of our patients with meningiomas. Undoubtedly, such displacement may occur, though probably infrequently, the tendency of such tumors being to "underlie and elevate both chiasm and arteries" (Cushing). It occurred in 3 of 17 cases reported by Tucker *et al.*,[15] 2 of the 3 being described as large, and only in 1 of the 9 cases of Bakay and Bean.[1]

Elevation of the supraclinoid part of the siphon occurred in 4 of our 7 cases; in none was it displaced laterally. Bakay and Bean[1] reported elevation of this vessel in 6 of 9 cases and lateral displacement of the bifurcation of the internal carotid artery in an equal number.

Elevation of the horizontal component of the anterior cerebral artery was observed in all of our cases. Bakay and Bean[1] reported this change in 5 of their 9 cases and Udvarhelyi *et al.*[16] in 2 of 7 cases; a "doubtful" elevation occurred in another 3 of the latter series.

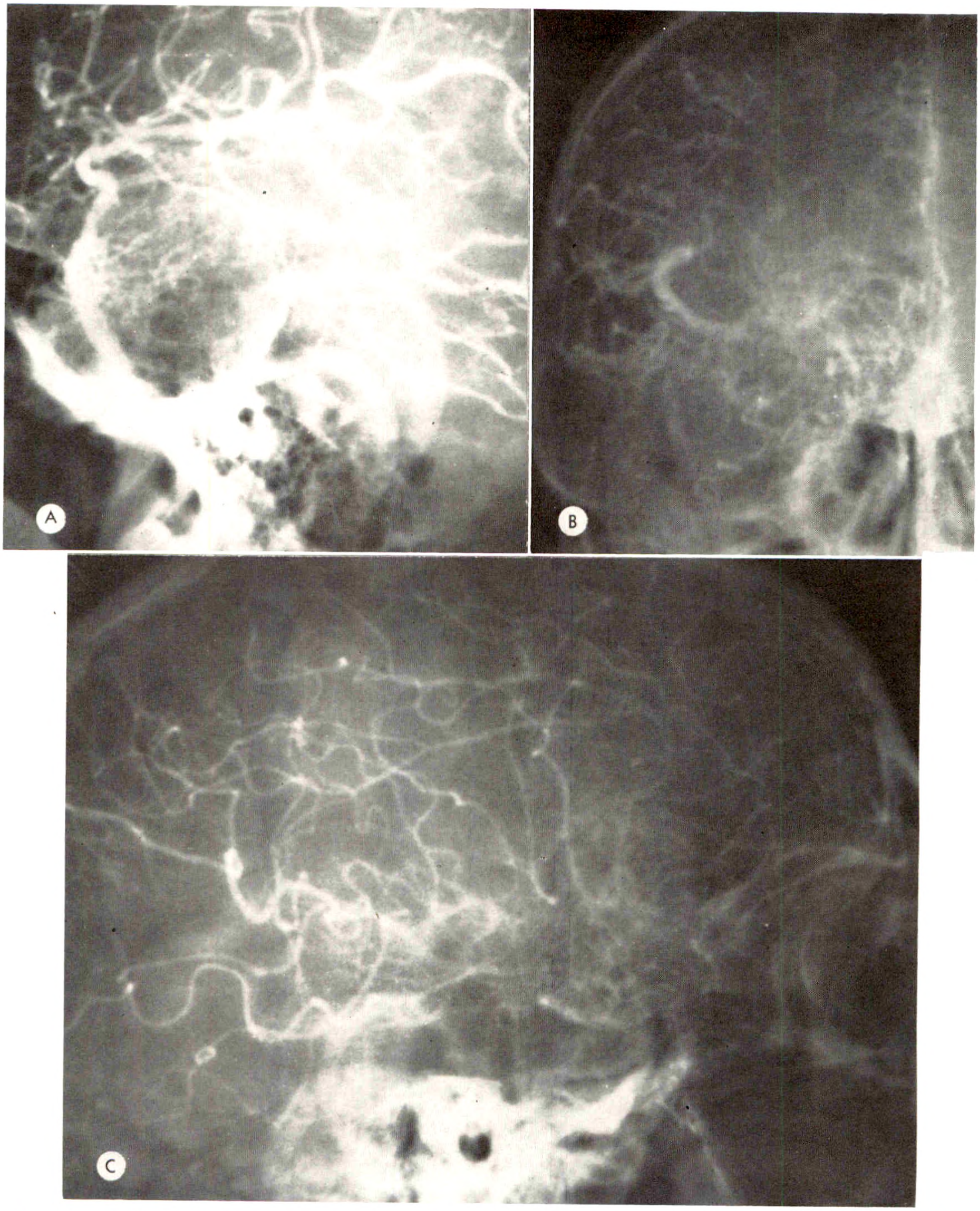

FIG. 14. (*A*, *B* and *C*) Pituitary adenoma with tumor "stains." *A* and *B* are lateral and anteroposterior projections of the same case.

Tucker *et al.*[15] made the following comments concerning the angiographic abnormalities in suprasellar meningiomas: "The horizontal components of both anterior cerebral arteries and the anterior communicating artery usually were elevated and displaced posteriorly—and the terminal portion of the internal carotid artery was often displaced laterally and its supraclinoid portion was straightened and appeared to be

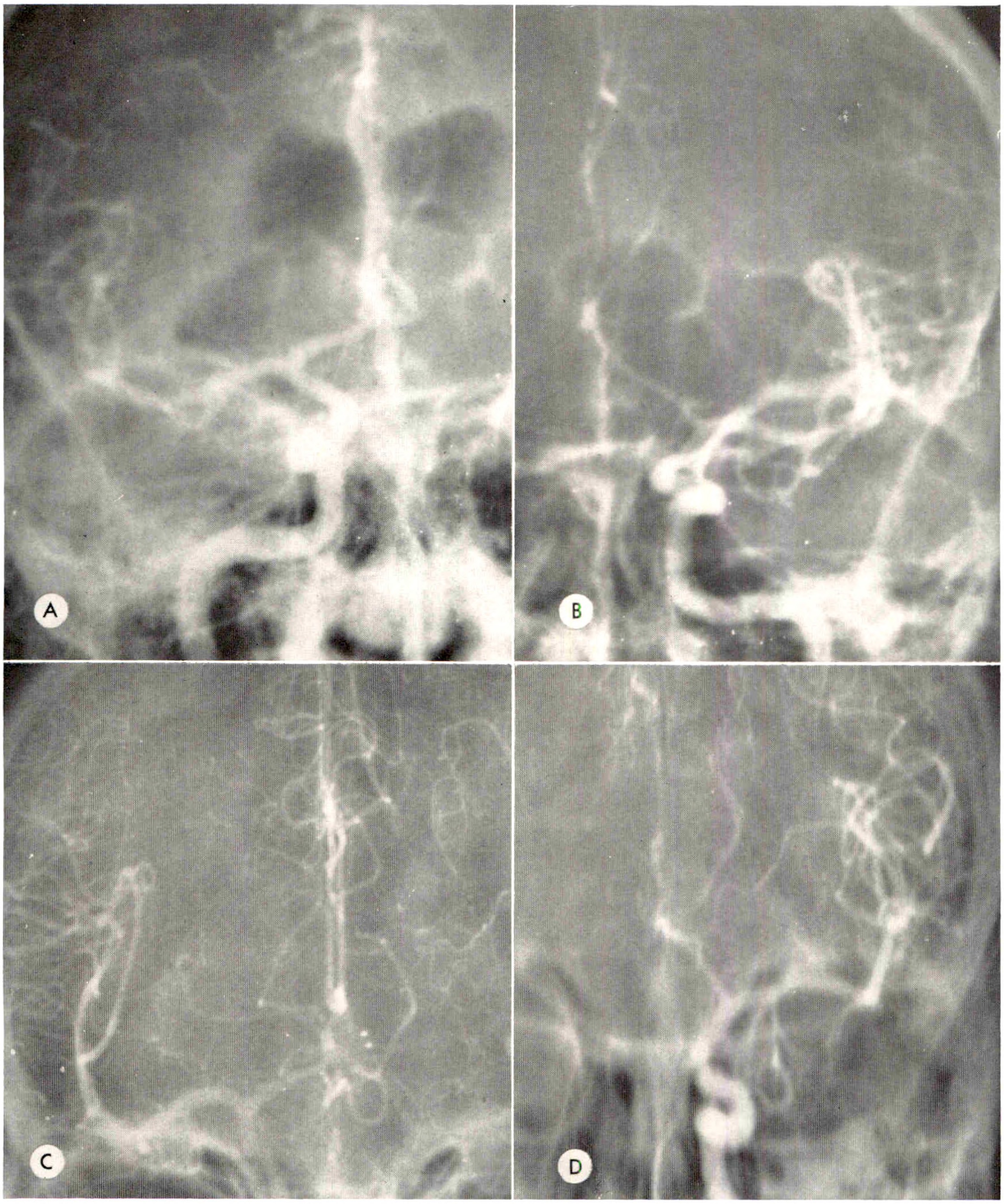

FIG. 15. (*A–D*) Appearance of anterior cerebral artery in cases of suprasellar meningioma. Bilateral arteriograms are represented in *A* and *B*, and *C* and *D*.

erect." Elevation of the basal vein and displacement of the internal cerebral vein posteriorly and superiorly were noted in 3 patients with large tumors.

Visualization of tumor vessels was reported in 1 case by Bakay and Bean[1] and in 4 cases by Tucker *et al.*[15]

In summary, angiographic abnormalities occur in a substantial proportion of cases of suprasellar meningioma, though the figures reported vary. As more experience is gained, undoubtedly angiography will as-

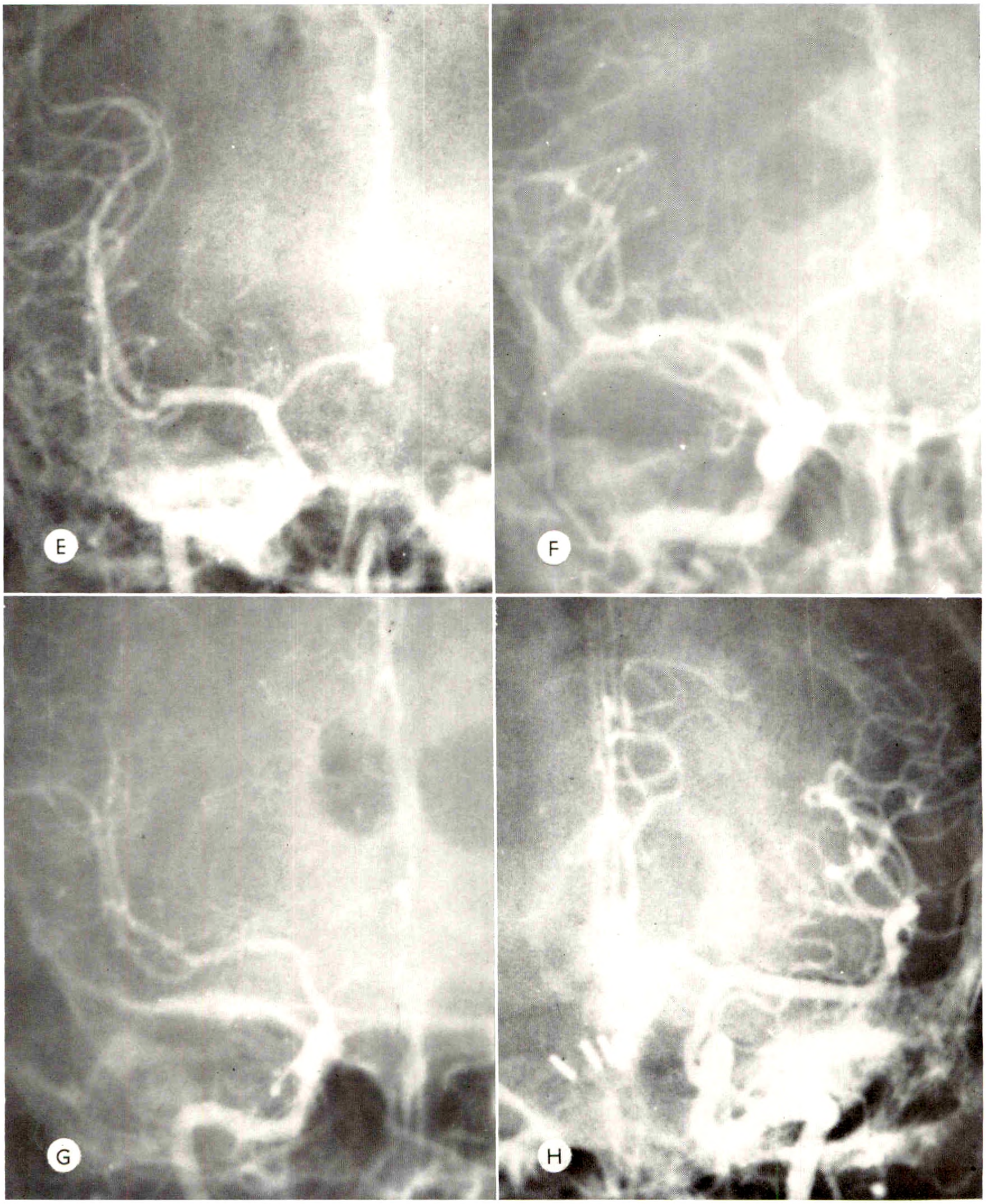

FIG. 15. (*E–H*) Appearance of anterior cerebral artery in cases of suprasellar meningioma. *H* is a postoperative arteriogram. Pneumoencephalograms were performed in most cases and residual gas is present in some of the films.

sume a more exclusive role in diagnosis. Difficulties are nevertheless bound to arise, especially when dealing with small tumors, in distinguishing between normal variants and lesser degrees of abnormality. When angiography fails to provide conclusive evidence of a tumor, further help must be sought through the use of pneumoencephalography. The most distinctive angiographic abnormalities in suprasellar tumors appear to be upward displacement of the horizontal segment of the anterior cerebral artery and elevation of the supraclinoid component of the internal carotid artery.

CRANIOPHARYNGIOMAS

Our experience with angiography in this type of tumor being limited to 2 cases, additional data were sought in the literature. Elevation of the first part of the anterior cerebral artery occurred in 4 of the 8 cases of craniopharyngioma included in the report of Udvarhelyi *et al.*;[16] in still another case the elevation was interpreted as "doubtful." In the experience of these authors, angiography was of greater diagnostic assistance in craniopharyngiomas (50 per cent) than in pituitary tumors (33 per cent) or suprasellar meningiomas (28 per cent).

Reporting their observations in 5 cases, Bakay and Bean[1] described upward displacement of the supraclinoid portion of the internal carotid artery in 2 and upward and lateral displacement of the posterior communicating, posterior cerebral and anterior choroidal arteries in 2; the horizontal component of the anterior cerebral arteries appeared either normal or slightly elevated. Bakay and Bean[1] concluded that angiography was of limited diagnostic value in

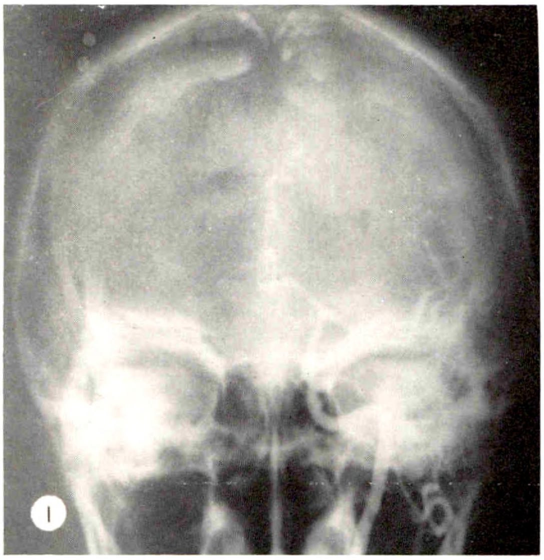

Fig. 15. (*I*) Appearance of anterior cerebral artery in a case of suprasellar meningioma.

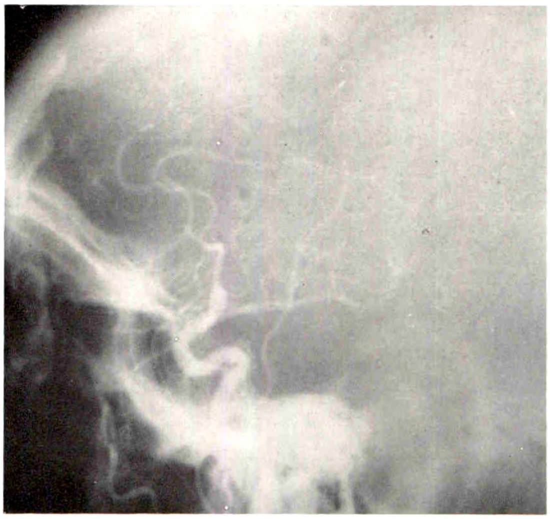

Fig. 16. Suprasellar meningioma with displacement of anterior cerebral artery superiorly and posteriorly.

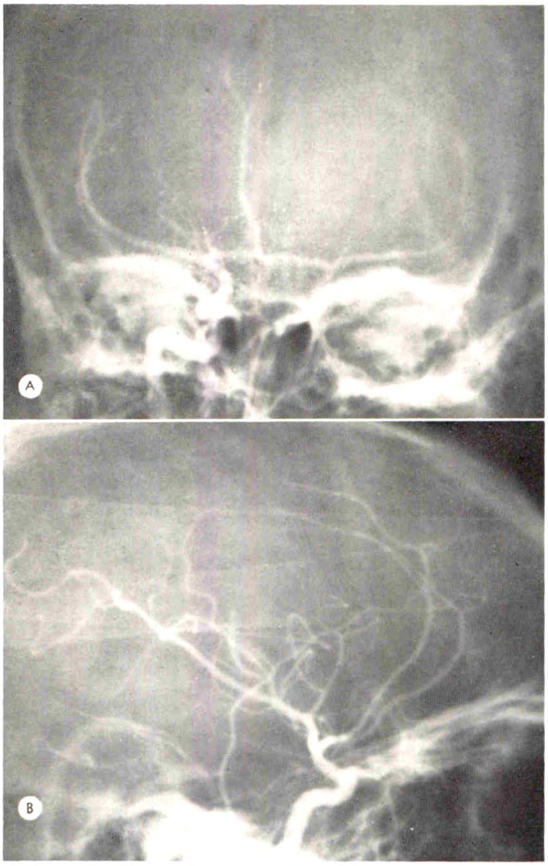

Fig. 17. (*A* and *B*) Craniopharyngioma showing evidence of hydrocephalus. The pericallosal artery is displaced from the midline.

craniopharyngiomas, the anterior cerebral artery being too far in front of the tumor to be affected by it. Suprasellar meningiomas, on the other hand, were more reliably outlined by angiography in their experience than any other type of suprasellar mass.

Displacement of the anterior cerebral artery upward and backward and stretching of the internal carotid artery represent the typical features of craniopharyngioma, according to El-Banhawy and El-Nadi.[7]

Judging from the diversity of the findings reported in a relatively small number of cases, it is difficult to come to any conclusion concerning the angiographic pattern characteristic of craniopharyngioma.

SUMMARY

The value of angiography in the diagnosis of tumors in the region of the sella turcica has been assessed on the basis of the authors' experience and reports in the literature. Though an important diagnostic adjunct, angiography is not invariably definitive in revealing the presence of a pituitary adenoma or suprasellar meningioma. The size of the lesion and its direction of growth are the determining factors in this respect. The problem is further compounded by difficulties which may arise in distinguishing between normal variants and lesser degrees of abnormality. Unequivocal changes establishing the diagnosis of a suprasellar tumor may be recognized in about one-third to one-half of the cases of pituitary tumor. Pathognomonic abnormalities also occur in a substantial proportion of cases of suprasellar meningioma, though the figures reported vary.

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PERCUTANEOUS RETROGRADE BRACHIAL ANGIOGRAPHY IN THE DIAGNOSIS OF ACOUSTIC NEURINOMA*

RESULTS IN 4 CASES

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FOR the past 2 years, vertebral angiography in this clinic has been performed by the retrograde injection of contrast medium into the brachial artery. This technique has proven so satisfactory that it is now routinely used in practically all patients in whom there is a clinical suspicion of vascular or neoplastic disease in the posterior cranial fossa. Since July, 1962, 4 patients with subsequently verified acoustic neurinoma have been investigated using this angiographic procedure. In each case, 3 of which showed normal internal auditory canals on skull roentgenograms, the diagnosis was confirmed by the angiographic visualization of small vessels arched over the surface of the tumor. It is well documented that acoustic neurinomas may be demonstrated by vertebral angiography and various angiographic findings in these tumors have been described.[3,8,9,11,14,18,21,23] It is not the primary purpose of the authors to reiterate these findings, but rather to emphasize that vertebral angiography performed by the retrograde brachial method is a practical means for demonstrating the presence of this tumor in questionable or atypical cases as well as to confirm the diagnosis and indicate the relative size and extent of the lesion in the more typical case of acoustic neurinoma.

METHOD

The patient is given 125 mg. sodium seconal intramuscularly approximately 45 minutes prior to beginning the procedure. He is placed on the roentgenographic table in a supine position and the skin about the antecubital area (usually the right side) is prepared antiseptically. The skin and subcutaneous tissue about the site of intended arterial puncture are infiltrated with 1 per cent procaine or xylocaine (lidocaine). A 17 gauge Grino-Cournand needle is inserted percutaneously into the brachial artery and threaded up the lumen of the vessel for a distance of 1 to 2 inches. Attached to the needle with Luer-Lok connectors is a short, flexible, nondistensible tube filled with saline. The needle and tubing are taped securely to the skin to prevent the system from becoming disengaged during the injection.

The injections are made with an Amplatz* CO_2 injector, using a pressure of approximately 225 pounds per square inch. A volume of 50 cc. of 50 per cent hypaque (sodium diatrizoate) is used for each injection. During the injection, a blood pressure cuff about the arm just below the needle is inflated to above systolic pressure to prevent flow of contrast medium distal to the brachial artery.

Serial roentgenographic exposures of the head are made using a Schönander film changer, the latter being adjusted so that two exposures are made each second for a total of 6 seconds. In the usual case, two injections are made which allow for serial roentgenograms in both, half-axial anteroposterior and lateral projections with a minimum of scattered radiation.

After the study is completed, the arterial

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* Amplatz injector—Model E.—Series No. 533, Neadmac Co., Inc., 5411 International Parkway, Science Industry Center, Minneapolis 27, Minnesota.

* From the Departments of Radiology and Surgery, Division of Neurosurgery, Duke University Medical Center and Veterans Administration Hospital, Durham, North Carolina.

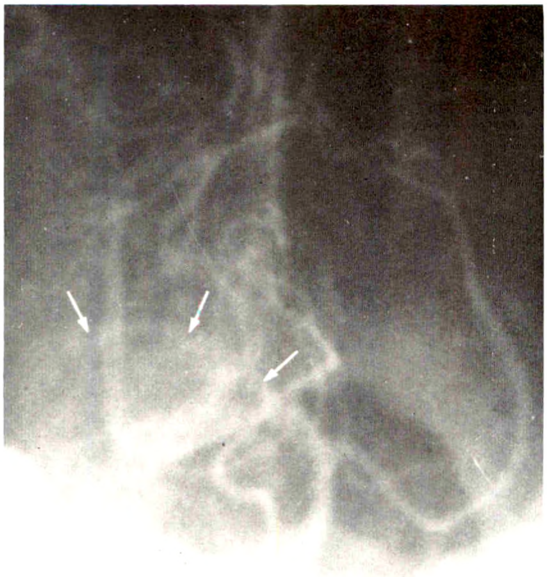

Fig. 1. Case i. Acoustic neurinoma, right (arrows).

needle is withdrawn and manual compression is maintained at the site of puncture for 5 to 10 minutes. If there is no further bleeding, the patient is returned to the ward and the radial pulse is checked every 30 minutes for 8 hours. The angiographic procedure requires about 1 hour for completion.

REPORT OF CASES

Case i. A 50 year old woman was admitted with a 3 year history of headaches and numbness over the right side of her face. The headaches were intermittent and diagnosed previously as atypical migraine. She noted "drooling" from the right side of her mouth one year prior to admission and had been "light-headed" on occasion but with no vertigo.

Examination. There was lateral gaze nystagmus to either side. Hearing was impaired to a slight degree bilaterally with bone conduction greater than air conduction on the right. An audiogram was not obtained. Moderate hypalgesia was present over all three divisions of the right fifth cranial nerve including an impairment in corneal sensitivity. Gait was slightly ataxic.

Lumbar Puncture. Opening pressure 90 mm. Cerebral spinal fluid protein 105 mg. per cent.

Skull Roentgenograms. Normal internal auditory canals.

Right Percutaneous Retrobrachial Angiogram, July 23, 1962 (Fig. 1). The upper portion of the basilar artery demonstrated a curvature with the convexity to the left. The right posterior cerebral artery was superimposed on the right superior cerebellar vessel; the left posterior cerebral artery did not fill. On the right side, a semicircle of small cerebellar vessels blended into the arc of the basilar artery and outlined a tumor which was situated above and medial to the right porus acusticus internus.

Angiographic Impression. Acoustic neurinoma, right.

Right Suboccipital Craniectomy, July 25, 1962. A large encapsulated tumor was totally resected from the right cerebellopontine angle.

Pathology. Neurinoma.

Case ii. A 49 year old woman was admitted with a 4 year history of intermittent tinnitus and unsteadiness of gait. Headaches and numbness over the left side of her face had been noted for 6 months. The headaches were described as dull and aching in character and located principally in the right occipital region, becoming more frequent and severe 1 month prior to admission. A gradual bilateral hearing loss was present for an unknown period of time and a diagnosis of Ménière's disease had been made elsewhere. Mild dysarthria and dysphagia had developed during the past month.

Examination. The gait was moderately ataxic. There was nystagmus on lateral gaze in either direction and mild hypesthesia over all three divisions of the left fifth cranial nerve, with an absent corneal reflex on the left side. Taste sensation was lost over the anterior two-thirds of the tongue on the left side. Mild dysarthria and minimal dysmetria of the left arm and leg were present.

Lumbar Puncture. Normal pressure. Cerebral spinal fluid protein 108 mg. per cent.

Skull Roentgenograms. Normal internal auditory canals.

Right Percutaneous Retrobrachial Angiograms, October 15, 1962 (Fig. 2, *A* and *B*). The basilar artery was vertical and slightly to the right of the midline. There was nonfilling of the left posterior cerebral artery. The left superior cerebellar artery was in a higher position than the one on the right and demonstrated a slight reversal of the normal caudal convexity. Concentric arcuate vessels were visible on the left, stretched over a tumor adjacent to the left porus acusticus.

Angiographic Impression. Acoustic neurinoma, left.

Left Suboccipital Craniectomy, October 17, 1962. Complete resection of a large tumor in the left cerebellopontine angle which had extended upward to the tentorium and downward to displace the ninth and tenth cranial nerves was performed.

Pathology. Neurinoma.

Case III. A 57 year old man was admitted with a 6 year history of intermittent dizziness and tinnitus. Hearing loss in the right ear and generalized headache had been present for 8 months. The headaches had become progressively more frequent and severe.

Examination. Hearing was impaired on the right. Bilateral papilledema was present. Hypalgesia was found over all divisions of the right fifth cranial nerve, including a decrease in corneal sensitivity. There was mild ataxia of the right arm.

Lumbar Puncture. Cerebrospinal fluid protein 168 mg. per cent.

Skull Roentgenograms. Normal internal auditory canals.

Right Percutaneous Retrobrachial Angiograms, December 11, 1962 (Fig. 3, *A* and *B*). The basilar artery was vertical and midline. The right posterior cerebral and superior cerebellar arteries were slightly higher in position than those on the left, but were difficult to separate from each other and were somewhat obscured by simultaneous filling of the right internal carotid bifurcation. On the later anteroposterior half-axial angiograms, an eccentric and oval "cloud" of abnormal vessels was visualized medial to the right porus acusticus and extending far to the left of the midline.

Angiographic Impression. Acoustic neurinoma, right.

Right Suboccipital Craniectomy, December 12, 1962. A large tumor was found to be completely filling the right cerebellopontine angle. A complete resection was performed.

Pathology. Neurinoma.

Case IV. A 25 year old man was admitted with a 3 month history of intermittent nuchal and suboccipital pain, blurring of vision and slight unsteadiness in gait.

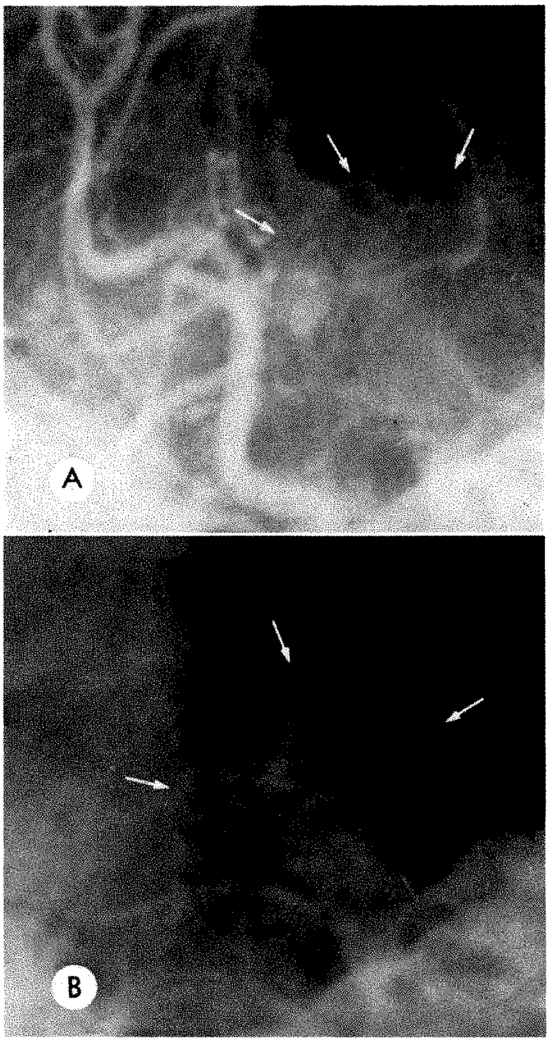

Fig. 2. Case II. (*A*) Acoustic neurinoma, left (arrows). (*B*) Tumor outline in late phase angiogram (arrows).

Examination. Bilateral papilledema was present. The remainder of the neurologic examination was entirely normal. Audiograms and caloric tests were normal.

Skull Roentgenograms. Slight widening of the right porus acusticus.

Right Percutaneous Retrobrachial Angiograms, February 1, 1963 (Fig. 4, *A* and *B*). The basilar artery was curved to the left of the midline. The right posterior cerebral artery was in a slightly higher position than the left one and had a somewhat flattened appearance in its anterior segment. The right superior cerebellar artery was arched upward in a definite reversal of the normal caudal curvature. A faint crescent

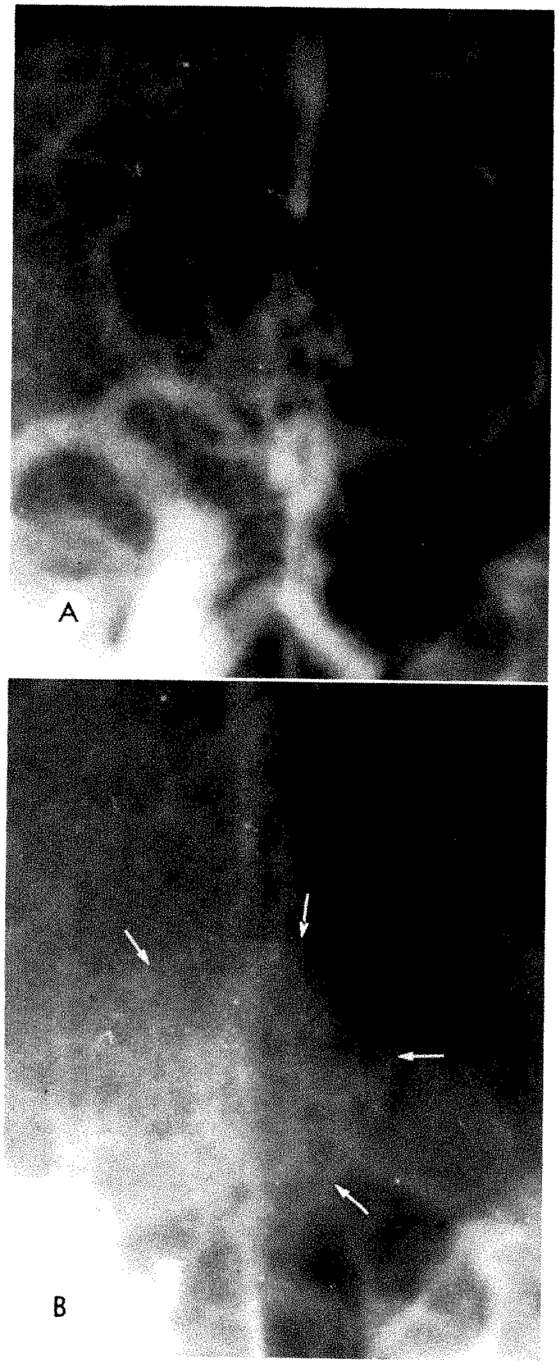

Fig. 3. Case iii. (*A*) Simultaneous filling of right internal carotid and vertebrobasilar distribution. (*B*) Acoustic neurinoma outlined in late phase angiogram (arrows).

of small cerebellar vessels was visible on the later phase anteroposterior angiograms. This vascular oval extended medially just to the left of the midline.

Angiographic Impression. Acoustic neurinoma, right.

Right Suboccipital Craniectomy, February 4, 1963. Complete resection of a large encapsulated tumor in the right cerebellopontine angle was performed.

Pathology. Neurinoma.

DISCUSSION

The angiographic findings in acoustic neurinomas have been described previously by a number of investigators, using a variety of techniques for obtaining vertebral angiograms. In 1937, Shimidzu[21] studied 3 unoperated, autopsied cases of acoustic neurinoma with respect to the vascular changes associated with this tumor. He described the following postmortem changes: (1) the posterior communicating artery was larger and the cerebellar vessels had more branches on the side of the tumor; (2) the blood supply for the tumor was derived from the internal auditory artery or from a branch of the anterior inferior cerebellar artery; and (3) the first part of the middle cerebral artery was elevated by the uncus, which had been displaced upward and forward by the tumor pushing through the tentorium. Shimidzu carried out vertebral angiography by percutaneous subclavian injection in 3 cases of acoustic neurinoma and found that the angiographic findings equaled those on postmortem examination. He did not include roentgenograms showing the direct outline of the tumor by angiography. Sjöqvist,[23] using an "open" method of vertebral angiography, demonstrated lateral displacement of the basilar artery in a patient with a cerebellopontine angle tumor but concluded that this finding could not be considered diagnostic since another case was studied with a similar displacement of the basilar artery and no tumor was found at operation. Radner[18] introduced the technique of vertebral angiography by catheterization of the radial artery and included in his initial report the anteroposterior angiogram of a verified acoustic neurinoma which showed a curved displacement of superficial vessels around the involved porus acusticus. Lindgren,[9] who introduced a direct percutaneous method of vertebral angiography in 1947, pointed out normal variations in the course of the posterior cerebral and superior cerebellar arteries in the anteroposterior angiogram and emphasized the similarity of these variations to those changes usually found in an acoustic neurinoma. Radner,[10] in 1951, amplified his original report and included 10 cases of verified acoustic neurinoma studied with his technique. He noted displacement of the basilar artery in 2 cases and tumor vessels in 2 other cases. Olsson[11] reported 14 cases of acoustic nerve tumors with vertebral angiograms obtained with the technique described by Radner. In 10 of his cases, the basilar artery was shifted away from the midline, but he felt that this finding was of limited value due to the marked variations in the course of this vessel in normal cases. The posterior cerebral artery took a "flattened" course on the affected side in 8 cases and the superior cerebellar artery on the side of the tumor was described as abnormal in all cases, with a marked "cranial flattening" in 13. Olsson found pathologic vessels in 9 cases, consisting mainly of fine arched vessels encompassing the tumor. Hauge[7] found positive findings on the angiograms of 5 cases of verified acoustic neurinomas. Displacement of the posterior inferior cerebellar artery and effacement of its loops were present in all cases, and in 2 of the cases "direct structural vascular changes" were visible at the tumor site, consisting of crescentic vessels outlining the tumor. Columella and Papo[3] carried out successful vertebral arteriography in a series of 220 cases. Included in this group were 19 patients with acoustic neurinomas and 5 with meningiomas in the posterior fossa. Without further analysis, they stated that "a pathological circulation" was observed on the arteriogram in 11 of these 24 cases (19 neurinomas, 5 meningiomas). Greitz and Lindgren[6] recently stated, "A tumor of the cerebellar pontine angle may cause a cranial and medial dislocation of the superior cerebellar artery. This is difficult to evaluate because of the numerous normal variations. It is easier to evaluate the dislocation of the smaller arteries, usually branches from the superior posterior cerebellar artery located on the surface of the cerebellar hemisphere adjacent to the inter-

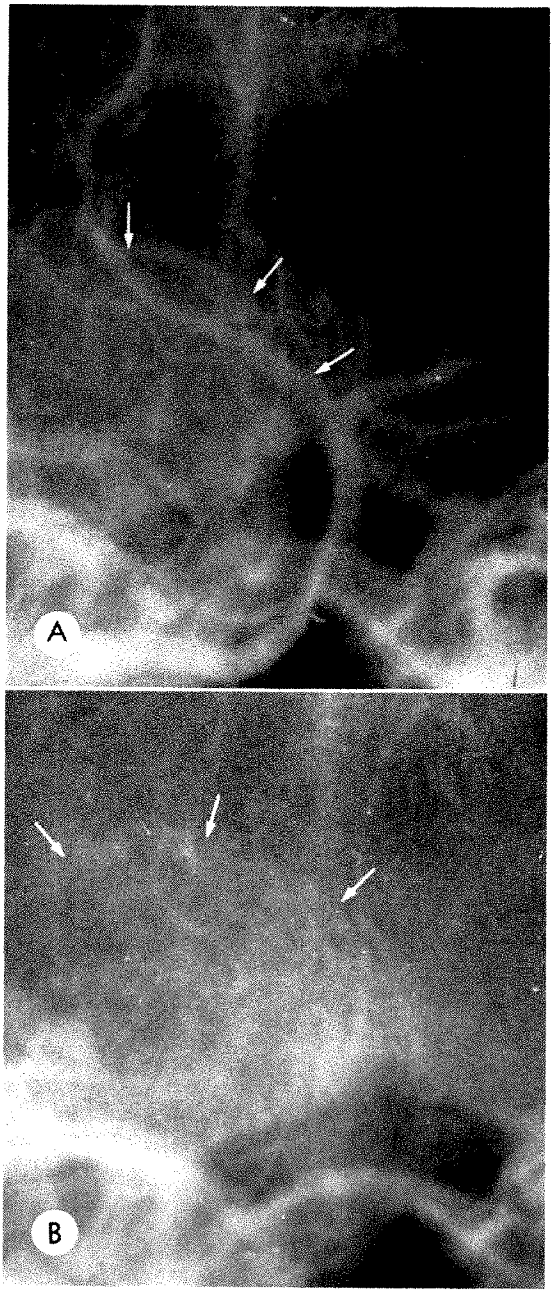

Fig. 4. Case iv. (*A*) Right superior cerebellar artery displaced upwards (arrows). (*B*) Acoustic neurinoma outlined in late phase angiogram (arrows).

nal auditory meatus. These vessels are stretched and may surround the tumor in an arc-shaped fashion. The small veins of the cerebellopontine angle may also be displaced in an arc-shaped manner away from the internal meatus."

Other authors[4,11,12,24] have reported cases of acoustic neurinomas which showed changes on the vertebral angiogram in keeping with those described by the authors cited above.

From the aforementioned, it is evident that the most significant single finding in the diagnosis of an acoustic neurinoma by vertebral angiography is the actual visualization of small vessels arched over the surface of the tumor. Although this direct vascular outline of the tumor has been, at best, an inconstant feature of other methods of vertebral angiography, this finding was present in each of our 4 cases in which the percutaneous retrobrachial approach was utilized. Serial roentgenograms in each phase of the posterior circulation enhanced the opportunity for outlining the tumor directly, and for this purpose the anteroposterior half-axial projection was the most valuable.

The clinical features in some cases of acoustic neurinoma are so characteristic that the need for special diagnostic studies such as vertebral arteriography or fractional pneumoencephalography is limited. However, cases are seen with atypical findings, and in this group contrast studies that serve to establish the diagnosis may be necessary. In the 3 cases in the present series with entirely normal internal auditory canals on skull roentgenograms, vertebral angiography was of considerable diagnostic aid, serving not only to affirm the diagnosis but to indicate the relative size and extent of the tumor as well.

The results of the present study indicate that vertebral angiography by retrograde injection of the brachial artery is a practical means of demonstrating the presence of acoustic neurinomas. The technique of vertebral angiography by retrograde brachial injection was first introduced in this country in 1955 by Gould, Peyton, and French.[5] They were able to visualize the vertebrobasilar system in 5 of 9 adults and in all of 7 children studied. Angiography disclosed a venous angioma in the posterior fossa in 1 patient and in another case there was evidence of hydrocephalus (due to a thalamic tumor), shown by stretching of the anterior cerebral artery in the lateral projection. They were not satisfied with the degree of contrast obtained with this technique in their adult group. In 1957, Collins, Slade, and Lockhart[2] reported their results with retrograde brachial angiography in 17 adults. Vascular lesions were demonstrated in 5 of their cases. These consisted of an aneurysm of the basilar artery in 1 case and in 4 instances arteriovenous malformation was noted. Angiography was not considered helpful in 5 cases of possible posterior fossa tumor. They concluded that the retrobrachial technique was a useful procedure in adults as well as in children. The cutdown method was used by both of these groups to cannulate the brachial artery.

Subsequent modifications of the procedure of retrograde brachial angiography by numerous workers, including the use of the percutaneous method for cannulating the brachial artery,[1,8,10,13,15–17,20,22] have established this technique as a relatively simple, safe and practical method for outlining the vertebrobasilar system and the internal carotid and its branches on the right side. In addition, the technique has proven a useful means for visualizing the entire extracranial portions of the right carotid and both vertebral arteries for the purpose of demonstrating segmental stenoses of these vessels.

Although considerable literature now exists on the technical aspects of the retrograde brachial method of angiography and the possibilities for outlining the extracranial and intracranial vessels, little has been written regarding the actual demonstration of neoplastic lesions in the posterior fossa with this technique. The indications from our experience with this procedure in various vascular and neoplastic lesions in the posterior fossa are that the degree of roentgenographic contrast obtained, in terms of diagnostic accuracy, is at least comparable with any other method for performing vertebral angiography. A report of our experience in the spectrum of posterior fossa disease is now in preparation. Although our present series of verified acoustic neurinomas studied by this technique is admittedly small, the consistent diagnostic accuracy obtained with this tumor is in itself evidence of the value of the retrograde brachial method of vertebral angiography.

SUMMARY

Four consecutive cases of acoustic neurinoma have been accurately demonstrated by the percutaneous retrograde brachial approach in vertebrobasilar angiography.

The relative simplicity and safety of this procedure, combined with positive results in this series, indicate that this method is a practical approach to the angiographic diagnosis of acoustic neurinoma.

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A CASE OF ANEURYSMAL MALFORMATION OF THE VERTEBRAL AND BASILAR ARTERIES CAUSING CRANIAL NERVE INVOLVEMENT*

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THIS case is presented because the patient's symptoms and signs were caused by an unsuspected malformation of the vertebral and basilar arteries which was demonstrated but not diagnosed by air encephalography. The true nature of the lesion was obvious following its visualization by retrograde brachial artery angiography.

REPORT OF A CASE

W.W., aged 44, had 4 admissions to the service of Dr. I. W. Ginsburg at Temple University Medical Center from December 9, 1958 to June 15, 1959.

His complaints were occipital headaches radiating to the left eye, dysphagia, left tinnitus, and occasional sharp pain in the lower jaw and ear.

He was in good health except for mild hypertension until he was involved in a severe automobile accident in June, 1954. After that, he was drowsy and confused for a period of 7 weeks, had multiple bone injuries and was hospitalized for 3 months. The hypertension worsened and in January, 1955, he experienced transitory motor aphasia. Shortly thereafter, he developed severe left sided tic-like face pain. He had alcohol blocks elsewhere in September and November of 1957, without facial anesthesia and developed transitory left peripheral facial paralysis after 1 of the procedures.

In January, 1958, the patient had a transient right hemiparesis and sudden relief from the tic pain which was replaced by a numb sensation on the left side of the face.

The examination in January, 1959 showed a blood pressure of 200/130. The pupillary reactions and corneal sensation were normal. The visual fields and external ocular movements were normal except for difficulty in closing the left eye. The eye grounds showed generalized attenuation of the retinal arterioles. There was decreased pain sensation in the third division of the left trigeminal nerve as well as an incomplete left peripheral facial paralysis. The uvula was in the midline. Gag reflex was decreased and the tongue was protruded slightly to the right.

The tendon reflexes were equal. There was no Hoffmann or Babinski sign. No loss of sensation or motor function in the extremities was noted. There was no cerebellar dysfunction. The blood Wassermann, urea nitrogen, blood cell count and urinalysis were normal. The spinal fluid pressure was 215 mm.; the fluid was clear with one cell and 99 mg. per cent protein.

Roentgenograms of the skull were negative. An electroencephalogram showed diffuse cerebral impairment. An electrocardiogram revealed myocardial changes of the coronary ischemic type. The vestibular function was normal and an audiogram disclosed bilateral high frequency nerve loss. Examination of the larynx showed a slight weakness of the left vocal cord. A barium swallowing study was normal.

Fractional air encephalography was performed on January 5, 1959, which showed a large fourth ventricle displaced to the left and

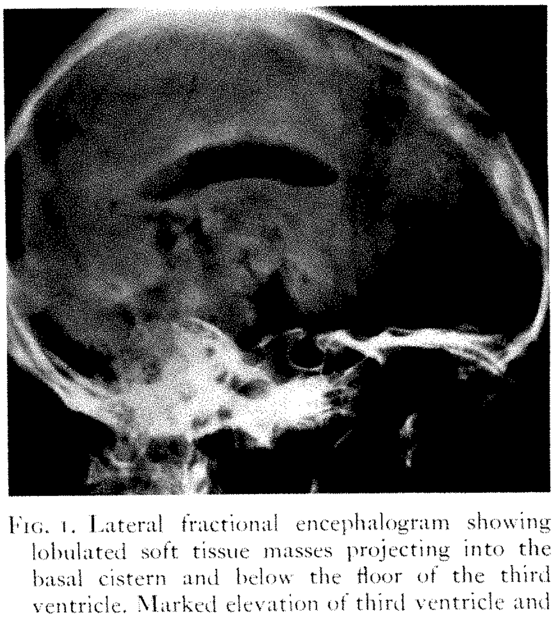

FIG. 1. Lateral fractional encephalogram showing lobulated soft tissue masses projecting into the basal cistern and below the floor of the third ventricle. Marked elevation of third ventricle and aqueduct is also present.

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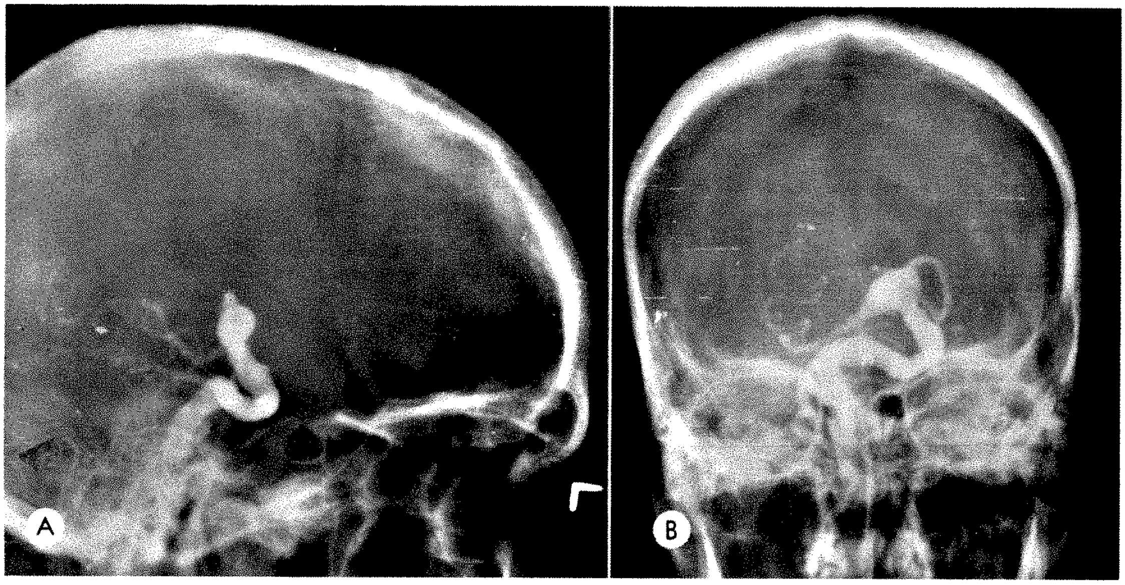

Fig. 2. (*A*) Lateral vertebral angiogram. (*B*) Left vertebral anteroposterior angiogram demonstrates marked irregular dilatation, elongation and tortuosity of the basilar artery and fusiform aneurysmal dilatation of the proximal portion of the left posterior cerebral artery responsible for the encephalographic changes demonstrated in Figure 1. (*B* reproduced with permission from *Archives of Otolaryngology*, 1964, *79*, 138–144. Copyright, 1964 by American Medical Association.)

encroached upon by a right-sided mass. The pontine cistern was decreased but there was good visualization of the interpeduncular and chiasmatic cisterns which, however, appeared to be encroached upon by lobulated soft tissue masses. The aqueduct and floor of the fourth ventricle were displaced posteriorly and superiorly. The third ventricle was elevated and a rounded soft tissue mass projected below it into the chiasmatic cistern region (Fig. 1). The lateral ventricles appeared mildly enlarged but were not displaced nor deformed. The evidence pointed toward an unusual lobulated, noninvasive space-occupying lesion in the region described.

The left brachial artery was exposed and a Robb cannula inserted. Roentgenograms were made at the completion of the rapid injection of 40 cc. of 50 per cent hypaque. These (Fig. 2, *A* and *B*) showed a large vertebral artery and a greatly enlarged and extremely tortuous and elongated basilar artery. The proximal portion of the left posterior cerebral artery also showed fusiform aneurysmal dilatation. The enlargement and tortuosity of the basilar artery and the left posterior cerebral artery were obviously responsible for the displacement of the fourth ventricle, aqueduct and third ventricle and for the lobulated soft tissue masses projecting into the cisterns as visualized in the encephalographic study.

COMMENT

The reported patient had symptoms and signs of involvement of the left 5th, 7th, 8th and 10th cranial nerves secondary to an aneurysmal malformation of the vertebral and basilar arteries. The relation of the severe head injury in June, 1954, to the onset of the neurologic abnormalities, which began in January 1955, is conjectural. Although the vascular malformation was present before the accident, the patient had no neurologic complaints until approximately 6 months after the severe head injury.

We were unable to find a case in which a similar lesion was demonstrated by air studies and angiography. Taveras[1] had reported such studies in a patient with an arteriovenous malformation of the temporal lobe.

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SUBDURAL EMPYEMA

REPORT OF A CASE DEMONSTRATING THE UNUSUAL ANGIOGRAPHIC TRIAD

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THE literature on the angiographic appearance of subdural empyema has been relatively sparce. Our purpose is to briefly review the angiographic findings in subdural empyema and to report a case with an unusual triad of angiographic findings, reported only once previously.

CLINICAL PATHOLOGY

From the patho-anatomic point of view, the term subdural empyema denotes a collection of pus between the dura and the arachnoid. On microscopic examination, it presents with a layer of exudate and organizing granulation tissue adherent to the inner surface of the dura with a similar accumulation of inflammatory tissue on the outer surface of the arachnoid. There is frequently evidence of neomembrane formation adjacent to the dura; however, membrane formation is not prominent on the arachnoid.[7]

According to Courville,[4] the exudate usually covers the convexity of the cerebral hemisphere and does not penetrate to the interhemispheral area. Schiller *et al.*[12] however, found that interhemispheral progression was an occasional complication.

The etiology is manifold and includes the following:[9] (1) infection from a penetrating wound; (2) acute sinusitis, particularly frontal sinusitis; (3) septic thrombophlebitis from any systemic focus; (4) sequela to purulent leptomeningitis; (5) an infected subdural hematoma (rare); and (6) otitis media with subsequent brain abscess and /or subdural empyema.

Although the first recorded observations on subdural effusions were published in 1886 by Ceci and Onetti,[2] the first clinical correlation of the symptom complex of subdural empyema was described by Kubik and Adams[8] in 1943. They found that all of their 14 patients had elevated temperature, headache, and a stiff neck. Half had convulsions. Decreased levels of consciousness and occasionally focal signs such as aphasia or hemiparesis were elicited. Otitis media was detected in 12 of their 14 patients. The other 2 had roentgenographic evidence of frontal sinusitis.

Lumbar puncture shows slight increase in white blood cells and protein in the cerebrospinal fluid.

ROENTGENOGRAPHIC DIAGNOSIS

With clinical symptoms, the finding of sinusitis, particularly frontal,[1,11] or sclerosing mastoiditis[9] is contributory evidence. Encephalography may show a mass lesion over the cerebral hemispheres with no air in the subarachnoid space.[10] Depression of the lateral ventricles and deformity are occasionally seen and may be signs of interhemispheral extension.[10]

Angiographic findings similar to the findings in subdural hematoma have been described.[5,13] If the hematoma is acute and lies over the parietofrontal region, a lenticular shaped zone of avascularity immediately adjacent to the inner table in the parietal and angular distribution of the middle cerebral artery will be seen in sagittal view. Chronic accumulation will show the avascular zone to be semilunar in shape with the convexity pointing toward the brain surface. Infratemporal hematomas, acute or chronic, will show elevation and/or medial displacement of the middle cerebral artery.

Feiring *et al.*[6] and Weber,[14] in separate

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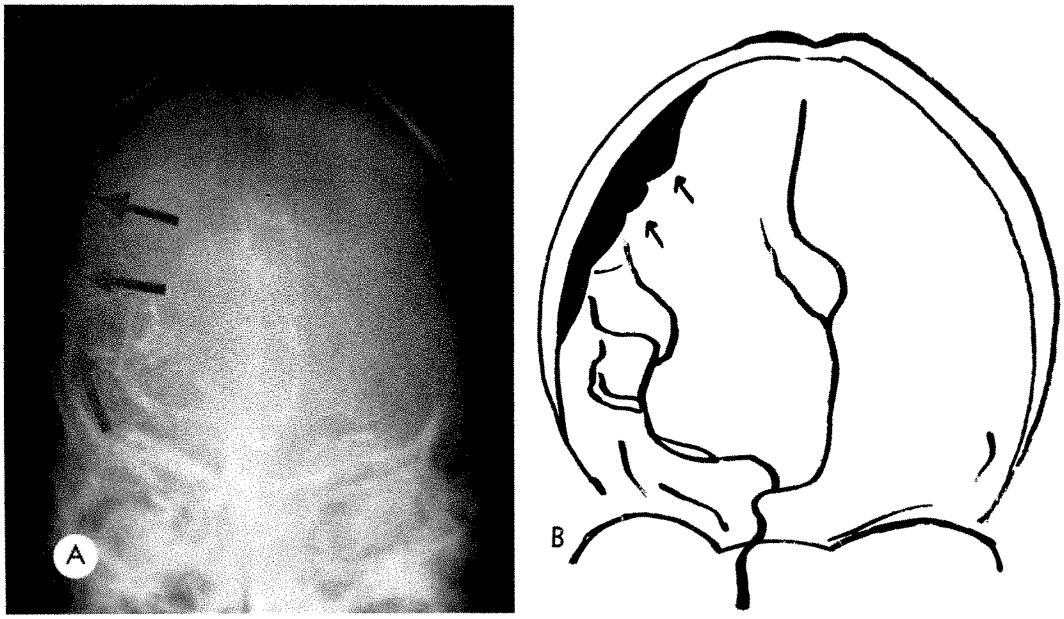

Fig. 1. (*A*) Right carotid angiogram. Sagittal view. Marked shift to the left of anterior cerebral (pericallosal branch) artery. The middle cerebral artery in its middle portion is elevated. Note medial displacement of knee and sylvian point. Arrows point to the irregular avascular zone adjacent to the inner table in the parietal and angular arteriographic distribution. (*B*) Diagrammatic sketch of *A*. Shaded area indicates the irregular avascular zone.

reports, have described a vascular pooling in the wall of the empyematous collection on the lateral cerebral angiogram. Furthermore, the lateral angiograms in these 2 reports have shown an avascular area, semilunar in appearance, with the convexity pointing towards the brain surface. Cornelis *et al.*,[3] reported a case in which the above two findings were present. There was, however, a third finding which was described as "an irregular image, festooned by the detachment, contrasting with the regular image obtained in the axial view in subdural hematoma." This triad, *i.e.*, an irregular detachment from the inner table of the calvarium on angiography, an avascular zone in the lateral view, and a vascular pooling in the wall of the empyema collection, would seem quite specific for a subdural empyema.

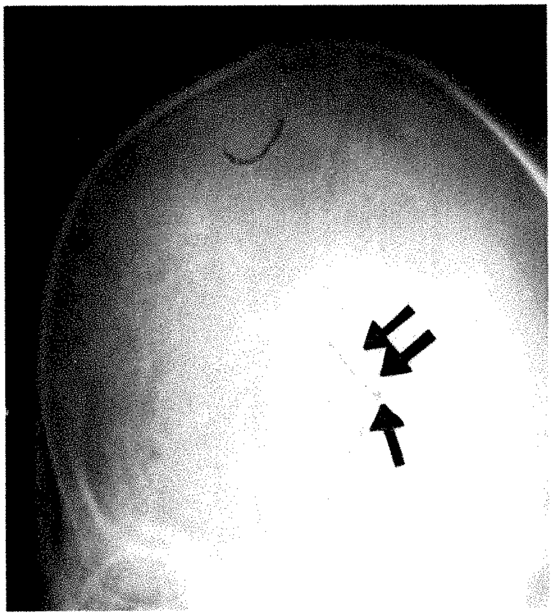

Fig. 2. Right carotid angiogram. Sagittal view. The venous phase shows marked displacement of the internal cerebral vein (single arrow), and an acute striothalamic-internal cerebral vein angle (double arrow) indicates a small right ventricle (touched-up).

REPORT OF A CASE

R.O., Hospital #2008479, a 29 year old white male, was admitted to the Boston City Hospital, April 8, 1964, in a semistuporous state.

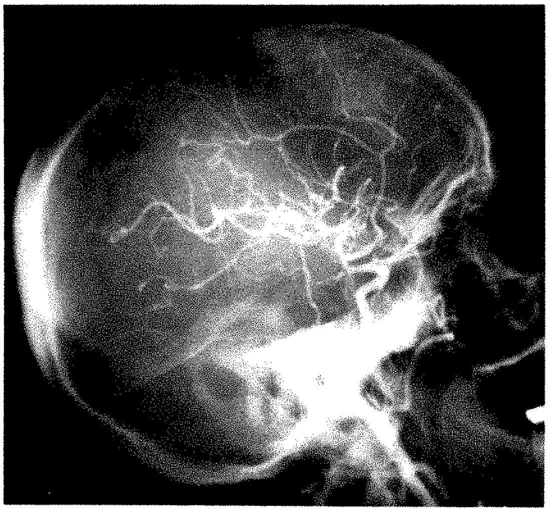

Fig. 3. Right carotid angiogram. Lateral view. Arteriographic phase showing demarcated zones of decreased vascularity in the anterior and posterior parietal regions.

Eight weeks prior to admission, an episode of frontal sinusitis was treated with oral penicillin. Five weeks before admission, he had a slight frontal headache. Two weeks before admission, the headache increased in severity and was associated with dizziness and blurring of vision in the right eye. Three days before admission, he complained of a very severe headache, vomiting, and progressive weakness of the left extremities.

Physical Examination. Temperature 100° F.; pulse 60/min; respiration 20/min; blood pressure 150/95. The patient was stuporous but oriented to time and place. His neck was stiff. The right pupil was less reactive to light than the left. There was a left facial weakness of an upper motor neuron type and marked weakness of the left arm and leg. The tendon reflexes were more active on the left. There was a Babinski sign on both sides and bilateral ankle and knee clonus. Clinically, subdural or epidural abscess was suspected, and carotid angiography was done.

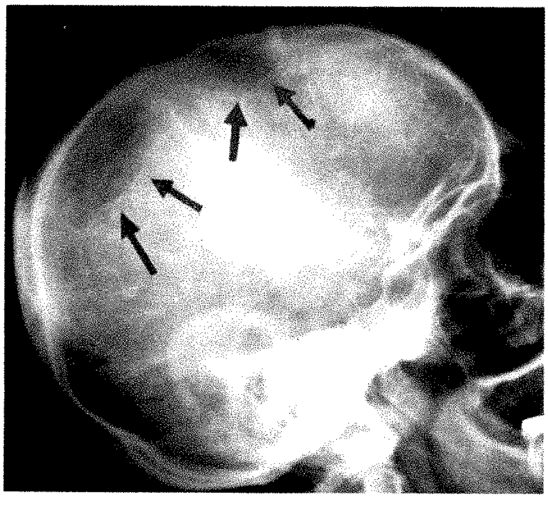

Fig. 4. Right carotid angiogram. Lateral view. The capillary phase shows two avascular zones (arrows) in the parietal region.

Findings on Right Carotid Angiogram (sagittal view). There was a marked shift to the left (1.9 cm.) of the pericallosal branch. The mid portion of the middle cerebral artery was elevated. The knee and the sylvian point of the middle cerebral artery were displaced medially. A scalloped 6–8 mm. zone of avascularity immediately adjacent to the inner table of the calvarium was noted in the region of the angular and parietal branches of the middle cerebral artery (Fig. 1, *A* and *B*). The internal cerebral vein was shifted 1.8 cm. to the left (Fig. 2). The striothalamic internal cerebral vein angle in the axial view was acute, thereby demonstrating a small ventricle on the right side (Fig. 2).

Findings on Right Carotid Angiogram (lateral view). The lateral arteriographic phase showed some diminished vascularity in the anterior and posterior parietal distribution (Fig. 3). The capillary phase disclosed two semilunar avascular zones, each measuring 4.5 cm. in length by 2.5 cm. in width, with the convexity pointing toward the brain surface (Fig. 4). Figure 5, a close-up view, shows the avascular zones to better advantage, and, also, the vascularized

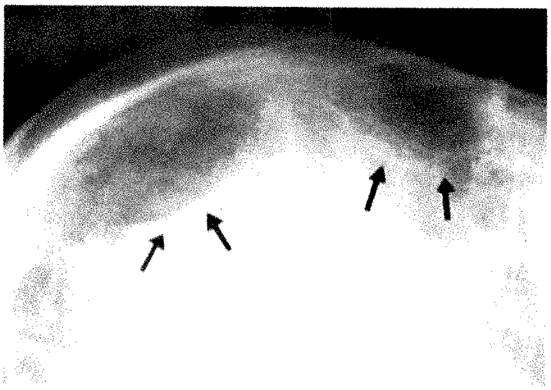

Fig. 5. Right carotid angiogram. Lateral view. Close-up view of Figure 4 showing the vascularized walls of the empyematous collections (arrows).

wall of the empyema collection. The late venous phase showed the persistence of the semilunar avascular zones with vascularized margins (Fig. 6).

Impression. The findings of the irregular clear zone in axial view, the elevation of the mid portion of the middle cerebral artery, the medial displacement of the knee and sylvian point of the middle cerebral artery, along with the avascular zones with vascularized walls in the lateral view, were consistent with subdural masses, most likely empyema. The areas of accumulation extended from the right parietal region over the surface of the brain and inferiorly to a right infratemporal location.

Hospital Course. Immediately following angiography, operation was performed under general anesthesia. Two burr holes were made 2.5 cm. to the right of the middle line, in the posterior frontal and mid-parietal region, exactly above the lesions demonstrated on angiograms. The dura was very tense, and after it was opened, about 25 cc. of pus escaped from each burr hole under great pressure. Then, the right temporal area was explored through a temporal craniectomy, disclosing a large accumulation of pus (50 cc.) in the subdural space. For the purpose of drainage and injection of antibiotics, soft Penrose drains and rubber catheters were placed in the burr holes and craniectomy. The wounds were closed loosely with a single layer of wire.

A direct smear of pus showed large numbers of streptococci and, on culture, the organism proved to be a nonhemolytic micro-aerophylic streptococcus, sensitive to penicillin. The day after operation, the patient was fully awake, but slightly confused. No other abnormal findings were noted apart from mild left hemiparesis. He was treated with penicillin (2,000,000 units every 2 hours) intramuscularly, and by local irrigation through catheters. Over the next 8 days, he made a slow, steady improvement, but continued to have an irregular fever ranging from 100–102° F. In the first few days, there was marked drainage of pus from the subdural space. The drains were removed on the 9th postoperative day. On the 14th day, the patient's condition suddenly deteriorated. He became very lethargic, confused, and developed signs of increased intracranial pressure. A revision of the sites of operation revealed marked brain swelling, but no new accumulation of pus. Right sided angiography was again performed.

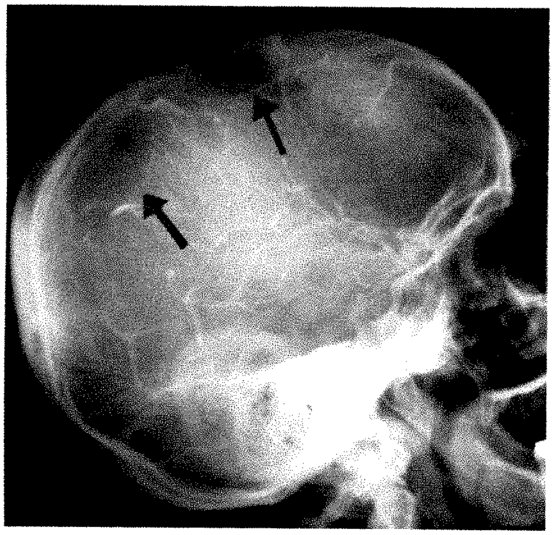

Fig. 6. Right carotid angiogram. Lateral view. The late venous phase shows the persistence of the two described avascular zones (arrows).

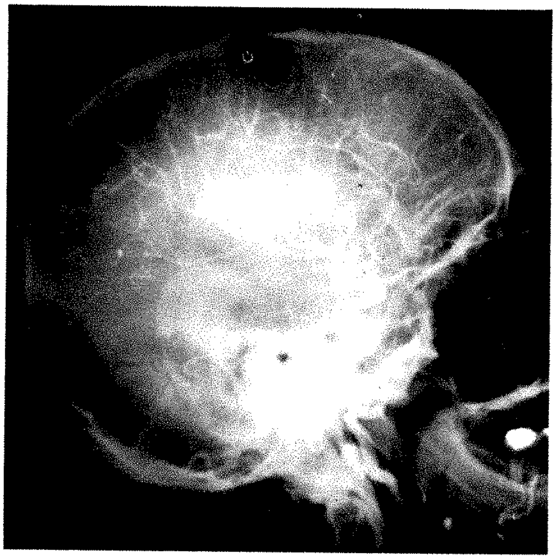

Fig. 7. Right carotid angiogram. Lateral view. This study, done 14 days after drainage of the empyema collections, shows the disappearance of the previously described avascular zones.

Findings on Postoperative Right Carotid Angiogram. There was a shift of the anterior cerebral (pericallosal branch) artery across the midline (1.5 cm.). There appeared to be no localized mass, and it was felt that cerebral swelling, probably secondary to cortical thrombophlebitis, was the mechanism. Of interest was the fact that the lateral angiogram (Fig. 7) showed disappearance of the previously described avascular zones.

Continuation of Hospital Course. An attempt was made to reduce the intracranial pressure through ventricular drainage. The left lateral ventricle was tapped through the frontal burr hole, and a rubber catheter was left in the ventricle for 3 days so as to allow drainage of the cerebrospinal fluid. Thereafter, the patient's condition again improved and he gained steadily. He became alert, fully oriented, and afebrile. The antibiotics were discontinued on the 25th day. He had been given a total of 240,000,000 units of penicillin.

On May 15, he appeared to be in excellent health. There was a bony defect in the right temporal area which pulsated. Neurologic examination was entirely negative.

DISCUSSION

The 3 major angiographic findings which we feel are specific for subdural empyema are: (1) a vascular pooling in the dural wall, seen in the frontal and/or lateral projections; (2) an avascular zone, semilunar in shape, seen on the lateral angiogram; and (3) an irregular detachment of vessels from the inner table seen in the frontal view. The reasons for these findings are as follows:

1. Vascular Pooling in the Dural Wall. This is due to the formation of an inflammatory neomembrane which may be seen in some cases of subdural empyema.[7] It is stressed, however, that there may be no membrane present. Under these circumstances, one would not expect to see a vascular pooling in the wall of the empyema collection.

2. Avascular Zone Seen on Lateral Angiograms. This roentgenographic finding can be explained by the presence of fibrinous and fibrous adhesions with loculation of an infected subdural fluid. The absence of a significant adhesive component would not then show these avascular sharply demarcated zones. It seems that the duration of the empyema is important in that an early process would be less likely to show this adhesive component and therefore be less likely to show these angiographic findings.[7]

3. Irregular Detachment Seen on Sagittal Angiograms. This was commented upon by Cornelis *et al.*,[3] and was quite striking in our case. The same reason advanced for the presence of avascular zones in the lateral angiogram would seem to apply as the patho-anatomic reason for the irregular stripping of the vessels from the inner table noted in the sagittal view. Again, loculation secondary to fibrinous or fibrous adhesions would need to be present. In an acute process, it can be readily seen that this appearance in the frontal projection could mimic an acute or chronic subdural hematoma.[5]

SUMMARY

The findings in a case of subdural empyema have been described. The irregular border of subdural empyema in contradistinction to the smooth wall of a subdural hematoma, the thickened vascular wall of the dura seen in the frontal and/or lateral projections, and the semilunar avascular zones noted in the lateral view should permit a diagnosis of empyema. We believe this to be the second reported case in the literature to embody this angiographic triad. The avascular zones with vascular walls have been described three times previously, and we believe that this represents the fourth such reported case.

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THE PREOPERATIVE DIAGNOSIS OF BRAIN ABSCESS BY THE ROENTGENOLOGIC DEMONSTRATION OF AN AIR-FLUID LEVEL*

A REPORT OF TWO CASES

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THE presumptive diagnosis of brain abscess may, on occasion, be quite tenable from the clinical picture in conjunction with supportive laboratory evidence, but a definitive diagnosis is rarely established in advance of exploratory surgery or postmortem examination. Moreover, the exact anatomic localization of an acknowledged brain abscess may be beyond definition even at the time of formal operation. It seems of value, therefore, to describe the fortuitous roentgenologic finding, whereby on two occasions, a definitive diagnosis of brain abscess as well as the delineation of its spatial relationships in brain tissue was established prior to surgical intervention. This present account concerns itself with 2 cases of intracerebral abscess in which the diagnosis was ascertained on the roentgenologic finding of an air-fluid level on plain upright roentgenologic study of the skull.

Roentgenographic methods have been, in a limited sense, the most successful single means in the identification of brain abscess to date, beyond such less specific clinical adjuncts as visual field perimetry and electroencephalography. However, the specialized roentgenologic studies which depend upon the intravascular injection of radiopaque contrast media or the introduction of air into the subarachnoid space or ventricular system are not without attendant hazards. Angiography is a valuable diagnostic method,[8] but, depending on the location of the lesion, the findings may be obvious or quite obscure.[1] Pneumoencephalography and ventriculography are likewise important investigative modalities, but not to be undertaken lightly. In contradistinction, the plain roentgenographic examination has been remarkably disappointing in its application to the problem of a suspected brain abscess. The findings of a shift in position of a calcified pineal gland or choroid plexuses, or the spreading of cranial sutures in children are infrequently noted, and only then in the presence of an aggressively expanding intracranial lesion. Rarely, calcification within the abscess or its capsule may be observed.[2]

The preoperative finding of an air-fluid level within a brain abscess on plain skull roentgenograms is an extreme rarity, probably because most roentgenologic examinations of the skull are conducted in the recumbent position. In the first reported case of brain abscess without air-fluid level diagnosed by ordinary roentgen methods, Glegg and Black[4] demonstrated a frontal lobe abscess by injecting as a contrast medium a substance known as Beck's bismuth paste through a fistula arising in a frontal sinus. The only documented case of otogenic temporal lobe abscess exhibiting an air-fluid level on plain roentgenograms was published by Eagleton[3] in 1922 in his textbook on brain abscess. An extensive review of the literature resulted in no additional cases in the absence of penetrating trauma. Russell and Taylor[7] in 1963 reported a case of intracerebral abscess with an air-fluid level due to *Clostridium welchii* following a penetrating injury. In adding their case to the antecedent literature, they acknowledge 16 cases of gas gangrene within brain substance, all due to the traumatic introduction of anaerobic bacteria, and within this group, 8 cases in which an air-fluid level was demonstrable on roentgenograms.

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The actual incidence of brain abscess has been markedly reduced by the modern advent of antibiotics but the problem of diagnosis has been compounded by the unfortunate masking effect of inadequate antibiotic therapy. The pathogenesis of brain abscess varies with any given series but approximately 10 per cent of cases are ascribed to frontal sinus origin, 40 per cent to otogenic evolvement and the residuum to such disease processes as compound craniocerebral trauma, congenital cardiovascular malformations and metastatic abscess from a thoracogenic suppurative focus.[2,6] As a sequel to an acute suppurative process, the etiologic microorganism in rhinogenic or otogenic brain abscess is usually staphylococcus, streptococcus or pneumococcus. In brain abscess due to chronic otitis media with cholesteatoma, the bacteria involved are usually the gram negative *B. proteus* or *Pseudomonas pyocyaneus*. However, other members of the coliform group, as well as various anaerobes may be incriminated in the chronic process; any of the mentioned gram positive cocci may also be found.

In considering the bacterial property of fermentation,[9] the beta-hemolytic streptococcus is considered the sole gas producing gram positive organism if lactose is available in the substrate. Due to its production of enzyme systems which inhibit the establishment of a fibroblastic barrier by the host, clinical streptococcal abscess with gas formation is probably a rare occurrence. *B. proteus* produces acid and gas from the fermentation of glucose and, to a lesser extent, of sucrose; it also produces ammonia from urea due to the action of urease. Coliform infections, especially *B. proteus*, produce large amounts of gas if the glucose concentration in the given substrate is increased, as occurs in diabetes mellitus with hyperglycemia.[5] *Pseudomonas pyocyaneus* does not produce gas from carbohydrate substrates, but yields only acid ferments. Anaerobic bacteria may produce gas in catabolic processes; *Cl. welchii*, for example, evolves a mixture of carbon dioxide and hydrogen from glucose, lactose and sucrose.

Roentgenographic examination of the skull in the following cases was conducted utilizing an upright head unit. The roentgen-ray beam is directed at more or less right angles to the vertical plane in all instances and this permits the demonstration of an air-fluid level should one be present.

REPORT OF CASES

Case 1. Admission #215293. A 16 year old white male was referred for the first time to the Manhattan Eye, Ear and Throat Hospital on December 15, 1955 because of profound swelling of the forehead which had been present and increasing for 3 weeks. Approximately 4 weeks prior to admission, the patient had suffered an upper respiratory tract infection which had rapidly progressed to an acute bilateral suppurative frontal sinusitis. One week after the administration of meager penicillin therapy and nasal medication from his local physician, he began to experience a severe frontal headache, which coincided with the onset of frontal swelling. Physical examination on admission revealed a lethargic, markedly toxemic young white male with a temperature of 104° F. There was a large fluctuant swelling in the frontal region extending from the glabella superiorly to the vertex of the skull and laterally to both parietal regions. There was a profuse purulent discharge from the middle meatus on both sides of the nose, more marked on the right. Moderate nuchal rigidity was in evidence. The remainder of the physical examination was unremarkable. The white blood cell count was 15,000 with a polymorphonuclear leukocytosis. A urinalysis was negative. Roentgen examination revealed an enormous subcutaneous abscess overlying the frontal bone and frontal sinuses with a large air-fluid level. There was also a 2.5 cm. lucency in the right frontal lobe (Fig. 1 and 2) with an air-fluid level. The frontal sinuses were cloudy and there was evidence of osteomyelitis in the anterior bony walls.

Under local anesthesia, incision and drainage of the subcutaneous forehead abscess was performed and almost a pint of pus was drained. The periosteum was incised and the frontal sinuses were opened bilaterally through their floors. Bacteriologic examination of the pus revealed *Staphylococcus aureus* and *Streptococcus viridans* on culture.

Over the next 24 hours, the patient's condi-

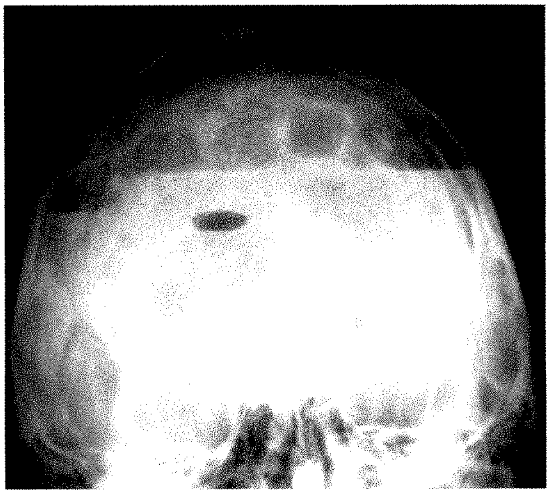

FIG. 1. Case 1. The frontal sinuses are large and cloudy. Multiple fluid levels are present in a massive subcutaneous forehead abscess. A small collection of gas and a fluid level are noted in the right frontal lobe.

tion continued to deteriorate with fluctuating levels of sensorium. He developed a bradycardia and a left Babinski sign was observed. Roentgenologic investigation of the skull was repeated (Fig. 3). A 2.5 cm. air-fluid level was still present in the right frontal lobe. A craniotomy utilizing a bilateral frontal sinusectomy approach was performed and osteomyelitis of the frontal bone was encountered. Both frontal sinuses were explored and the anterior and posterior bony plates of the frontal sinuses were found to be dehiscent near the mid-line. A fistulous communication draining purulent material was seen extending from the frontal sinuses through the dehiscence in the frontal bone to a large right frontal lobe abscess, which was evacuated of at least 40 cc. of pus. Osteomyelitic bone was excised and the cavity was exteriorized with packing. The patient improved steadily with parenteral penicillin and supportive therapy.

He continued to be well for over 15 months, and plastic repair of the frontal defect was contemplated. In late March of 1957, following another upper respiratory tract infection of 3 days' duration, the patient developed the clinical picture of a fulminating meningitis with recurrent generalized convulsions. The frontal region was surgically re-explored and a severe meningitis was encountered. No localized collection of pus was present; the abscess cavity previously described was well healed. The meningitis failed to respond to therapy with penicillin and the patient died within 24 hours.

Postmortem examination revealed free communication from the site of surgical drainage via the right frontal sinus to the right frontal lobe of the brain. The dura was normal above the defect. Thick white pus was present in the subarachnoid space overlying the superior

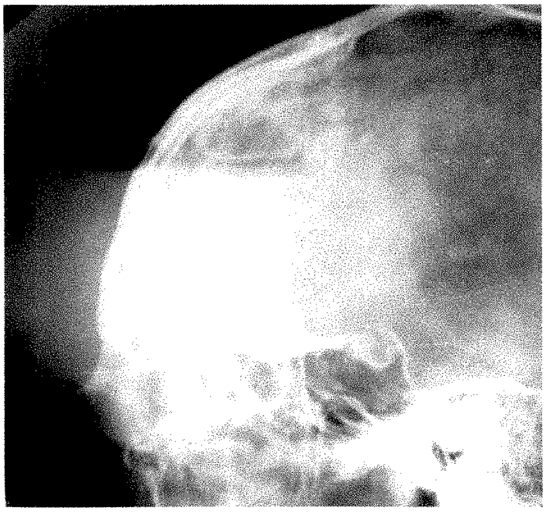

FIG. 2. Case 1. Lateral roentgenogram also reveals a large subcutaneous forehead abscess with a gaseous collection and a fluid level. A small fluid level and gaseous collection are noted in the right frontal lobe. The anterior bony wall of the frontal sinus is thinned and partly destroyed by osteomyelitis.

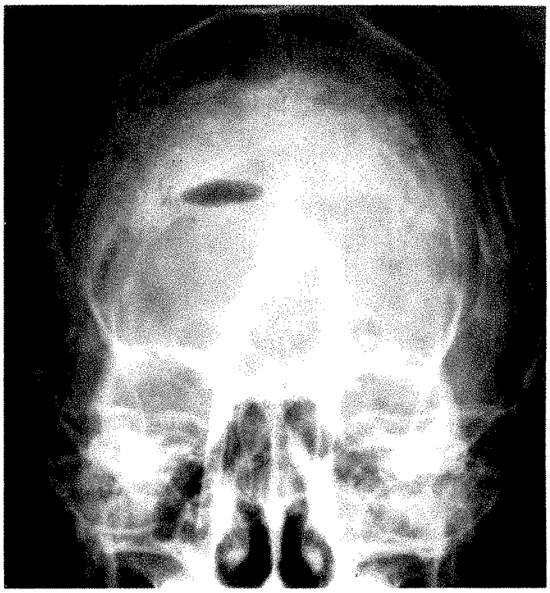

FIG. 3. Case 1. A small collection of gas with a fluid level persists in the right frontal lobe after drainage of subcutaneous abscess of the forehead and of empyema of the frontal sinuses.

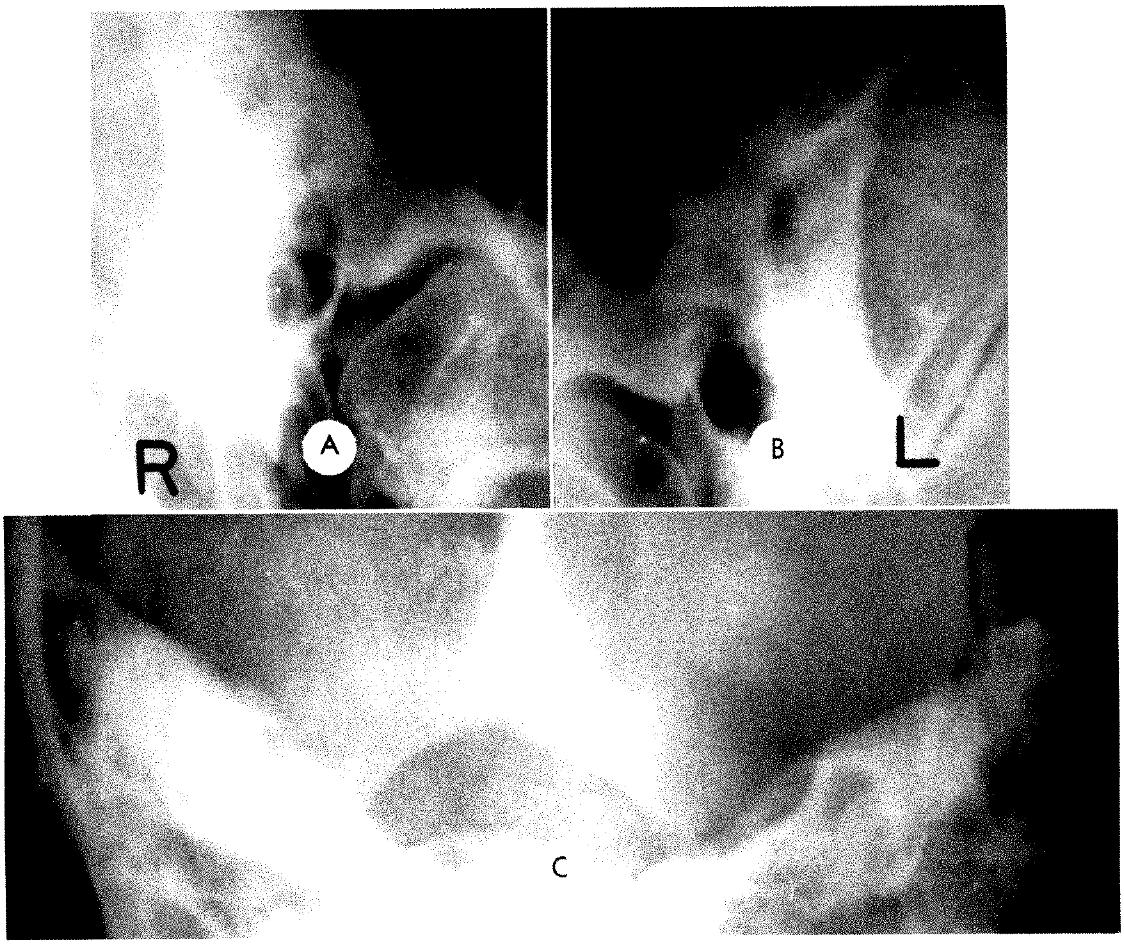

FIG. 4. Case II. (*A*, *B* and *C*) Roentgenograms of the right and left mastoids. Bilateral chronic sclerosing mastoiditis is demonstrated. Bone absorption is noted in the right epitympanic recess.

frontal sulci. The poles of both frontal lobes were softened but no localized collection of pus was found. A small amount of pus was present in the left nasofrontal duct.

Microscopic examination revealed acute inflammation and necrosis about the operative area. An acute purulent meningitis with thrombosis of vessels and focal involvement of the adjacent cerebral cortex was observed. Culture of the exudate within the subarachnoid space yielded *Staphylococcus aureus*.

CASE II. Admission #292030. A 38 year old Puerto Rican male was admitted to the Manhattan Eye, Ear and Throat Hospital on December 10, 1963 with a chief complaint of recurrent purulent discharge from the right ear and right mastoid pain for 7 days. The patient had a history of long-standing bilateral chronic otitis media in association with a repaired congenital cleft palate. At the time of admission he was afebrile and oriented. The right posterosuperior osseous canal wall was sagging and the tympanic membrane was obscured. There was minimal tenderness over the mastoid antrum. There was no evidence of nuchal rigidity. The remainder of the physical examination was unremarkable. The white blood cell count was 14,000 with a polymorphonuclear leukocytosis. Urinalysis was negative. Systemic antibiotic therapy was initiated.

Mastoid roentgenograms revealed a sclerotic right mastoid with pneumatization limited to the peri-antral area. These cells were cloudy and there was evidence of cell wall absorption in the attic-antral area (Fig. 4, *A*, *B* and *C*).

Over the next 3 days wide variations in temperature were noted. On the third hospital day the patient became somewhat drowsy and nuchal rigidity was in evidence. There were no cranial nerve palsies or papilledema, and the visual fields were full to confrontation. There

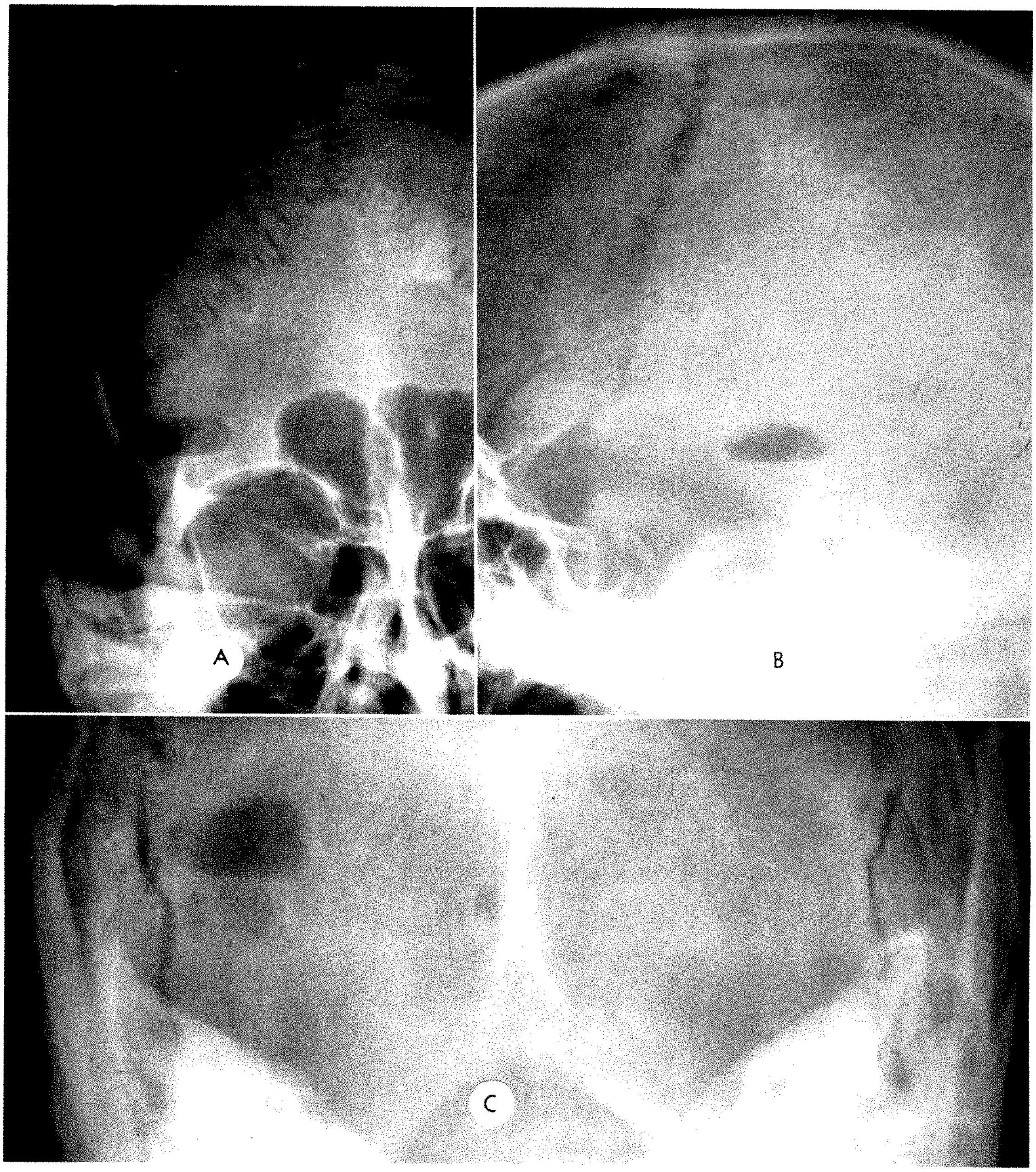

FIG. 5. Case II. (*A*, *B* and *C*) Gas and fluid level in the plane of the right temporal lobe are demonstrated in Caldwell, lateral and occipital views.

was no nystagmus. Motor examination revealed brisk symmetric reflexes and Chaddock's sign was elicited bilaterally. Upright skull roentgenograms demonstrated a 2.5 cm. air-fluid level in the region of the right temporal lobe (Fig. 5, *A*, *B* and *C*). The clinical and roentgenologic impression at that time was a right temporal lobe abscess secondary to a right chronic otitis media with cholesteatoma. Lumbar puncture was obviated by the roentgenographic findings.

Over the next few hours the patient's condition began to deteriorate with increasing headache, vomiting, restlessness and stupor. Under general endotracheal anesthesia a right radical mastoidectomy was performed via a postauricular approach. Spontaneous vertical nystagmus was observed in the operating room prior to the induction of anesthesia. At operation an attic-antral cholesteatoma was encountered. An extradural collection of pus was uncapped between the tegmen antri and middle fossa dura. The middle fossa dura was covered with granulations and a central area of prolapsing necrotic brain substance was visualized. Through this dehiscence approximately 30 cc. of foul creamy pus was obtained from a temporal lobe abscess. A catheter was inserted and the wound was left open.

The patient was treated with massive doses of parenteral chloromycetin and penicillin and the abscess cavity was irrigated with bacitracin solution. Intravenous urea was administered to reduce cerebral edema. Over the next 36 hours, the patient was semicomatose. There were no focal neurologic signs. Suddenly he experienced a grand mal seizure, following which he developed decerebrate posture and a dilated, fixed right pupil, and expired.

Postmortem examination revealed a large poorly encapsulated temporal lobe abscess lined with shaggy greenish exudate. The abscess was marginated by a diffuse cerebritis. There was evidence of fresh blood within the abscess cavity and in the ventricular system. There was no purulence within the ventricles, and the subarachnoid space was free of exudate. Culture of the pus at the time of surgery yielded *Pseudomonas pyocyaneus* and *B. proteus.*

DISCUSSION

In the first case reported, the organisms recovered were *Staphylococcus aureus* and *Streptococcus viridans*. The role of the *Staphylococcus aureus* is undisputed, but the *Streptococcus viridans* may have been present as a commensal. In any event, neither organism is an established gas producer in tissues. It is possible, therefore, that air was present in the abscess cavity for some other reason, such as the presence of free communication between the abscess and an air-containing paranasal sinus, or there may have been other undetected bacteria present, such as anaerobes. In the second case, it is most probable that gas was produced by the infecting *B. proteus*, although air could have entered through fistulous communication with the mastoid antrum. Hemorrhage from the abscess wall into the ventricular system probably precipitated death but presumably did not antedate the roentgenologic finding of an air-fluid level.

SUMMARY

Two cases of brain abscess definitively diagnosed by the preoperative roentgenologic finding of an air-fluid level are presented. This is a rarely observed occurrence, having not previously been reported in rhinogenic brain abscess and reported but once in otogenic brain abscess. The only additional air-fluid levels otherwise described have been in association with gas gangrene due to penetrating trauma. The diagnostic value of this sign is evident. It is suggested that it might be more frequently encountered if roentgenologic study of the skull were carried out in the upright position.

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ANGIOGRAPHIC DEMONSTRATION OF BILATERAL CAROTID BODY TUMORS*

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CAROTID body tumors are so rarely encountered that they are usually not considered when a patient presents with a mass in the neck. Only if angiographic study of the carotid vessels is performed, can a definitive preoperative diagnosis be made. We recently had the opportunity to study a case of bilateral carotid body tumors exhibiting the characteristic angiographic appearance of this neoplasm.

A review of the literature reveals that of the over 500 patients reported with this condition relatively few were studied by angiography. These all had characteristic roentgenographic findings. The angiograms of 15 of these patients have been published in the medical literature but only 3 of them are in American Journals.[13,16,17]

REPORT OF A CASE

R.S., a 57 year old female, was admitted to the Montefiore Hospital for the first time on February 20, 1962, with a chief complaint of pain and swelling of both sides of her neck. The patient had noted bilateral neck swelling for at least the preceding 25 years, with only slight increase in size. During the past year, the swelling became more pronounced, particularly on the right side and was associated with pain which radiated upward to her head. There had been a slight decrease in the power of her voice over the past few years but she had otherwise been in excellent health all during this period.

The patient was a well-developed somewhat obese female in no acute distress. Blood pressure was 120/70 mm. Hg in both arms. A fusiform swelling overlying the line of the sternomastoid muscle was noted bilaterally. The masses measured 6 cm. in diameter on the right and 3 cm. in diameter on the left and were nontender, firm and apparently pulsatile. They were movable laterally but not upward or downward. A systolic bruit was heard over both masses. Paresis of the left vocal cord was found on laryngeal examination. The remainder of the examination was unremarkable. The laboratory findings were all within normal limits. The ophthalmodynamometric study was interpreted as "consistent with decreased blood flow through both carotid arteries." Roentgenographic examination of the soft tissues of the neck revealed bilateral soft tissue densities laying anterior to C3–C5. These did not contain calcium or lipomatous tissue.

The patient was referred for roentgen study of the carotid vessels. Because of the bilateral involvement, it was decided to use the Seldinger[14] method of percutaneous transfemoral brachiocephalic arteriography. The catheter was placed in the aortic arch, 45 cc. of 75 per cent hypaque was introduced and serial roentgenograms in frontal and lateral projections were made. The angiograms revealed large vascular masses bilaterally (Fig. 1–4), intimately associated with the carotid bifurcations and fed mainly by branches from the external carotid arteries but also in part from branches of the subclavian artery via the thyrocervical trunk.

An exploratory operation was performed on the right side of the neck and a large vascular tumor completely encasing and attached to the carotid arteries at the bifurcation was found. Removal of the tumor was not felt to be possible because of its intimate relationship to the carotid vessels. A biopsy of the tumor was taken and tissue characteristic of a carotid body tumor was obtained. The patient made an uneventful recovery except for the occurrence of a twelfth nerve paralysis on the right side presumably secondary to the surgical intervention.

The patient was then referred for cobalt 60 radiation therapy and received a total tumor dose of 3,200 r delivered in 22 treatments over a period of 6 weeks through a large 14×14 cm. anterior neck portal. The tumors showed essentially no change in size following the course of irradiation and the patient was discharged from the hospital.

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The patient was readmitted to the hospital on September 29, 1963 with progressive dysphagia due to continued growth of the right-sided carotid body tumor with compression of the pharynx. It was decided that removal of the tumor would be necessary. This was performed by Drs. Som and Seidenberg with the use of an internal shunt in the carotid artery during the procedure. Because of the encasement of the carotid artery by the tumor, the vessel was injured but was repaired satisfactorily so that resection was not necessary. There were no neurologic complications following surgery. Six months following the procedure the patient was well without return of symptoms or tumor.

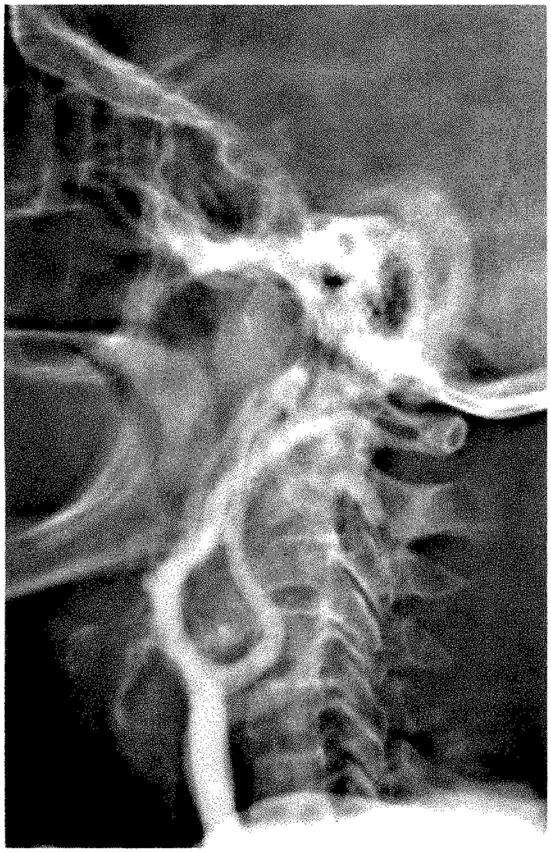

Fig. 1. Spreading of the carotid bifurcation bilaterally (two sides superimposed) with early "tumor stain" within the bifurcation can be seen.

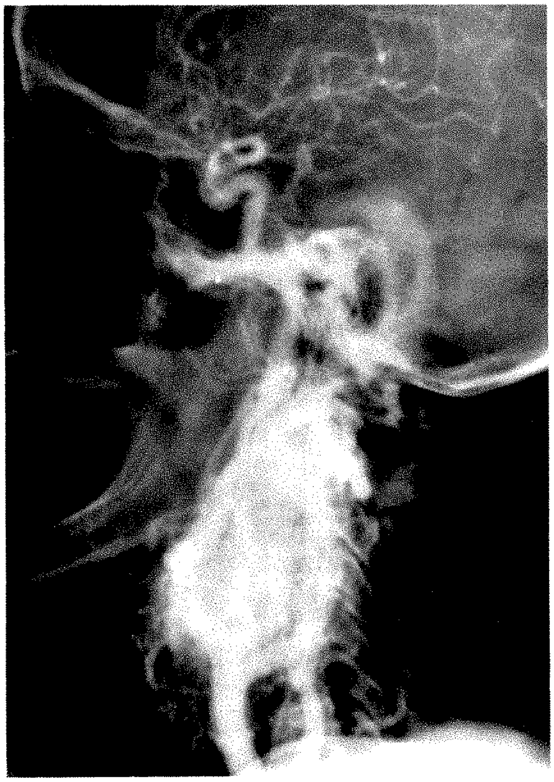

Fig. 2. The carotid bifurcations are completely surrounded and encased by a large vascular tumor.

DISCUSSION

The carotid body is situated at the bifurcation of the common carotid artery and is part of the chemoreceptor system which regulates blood pH, oxygen and carbon dioxide tension. Tumors of this group (which include the aortic, jugular and vagal bodies) are called chemodectomas and are histologically identical. The carotid body tumor is the most common. These tumors are almost always benign (although a number of metastasizing malignant tumors has been reported[2,11,15]), grow very slowly, and are usually asymptomatic. Occasionally, they enlarge sufficiently to press on the pharynx and cause difficulty in swallowing. They have also been reported to wrap around and compress the internal carotid artery. They rarely cause the carotid sinus syndrome.

The diagnosis of this tumor may be suspected on clinical grounds. A characteristic sign is that it can be moved freely from side

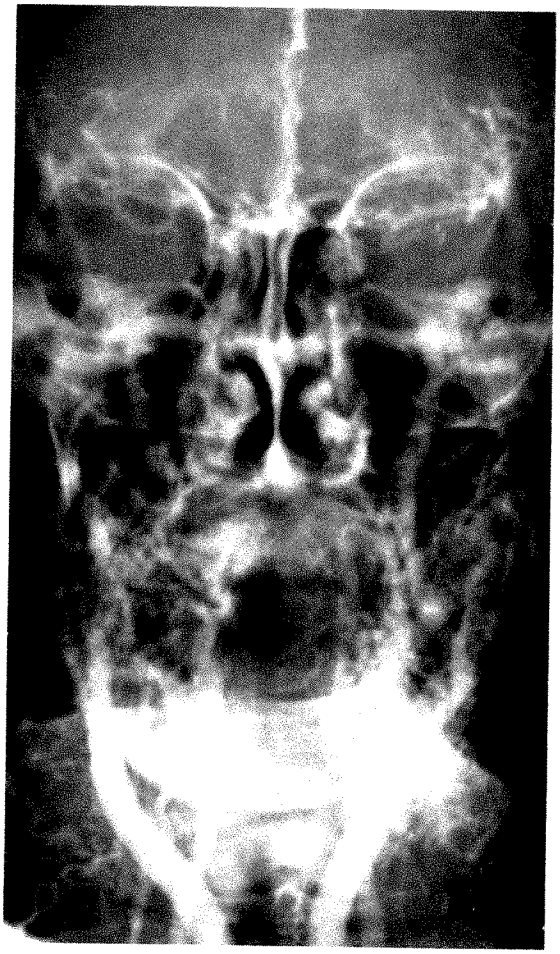

FIG. 3. Frontal angiogram demonstrates the lateral displacement of the carotid vessels and the extent of tumor.

to side but cannot be moved upward or downward. A definitive nonoperative diagnosis can be made only by angiography. In the study of atypical mass lesions in the neck, direct puncture of the common carotid artery as low as possible on the involved side is the simplest procedure. Percutaneous transfemoral or transaxillary passage of a catheter to the aortic arch with visualization of all the brachiocephalic vessels (as was done in this case) may also be performed. This latter technique is particularly indicated when bilateral masses are present and in cases when a unilateral mass is quite large and a low carotid puncture would be technically difficult.

The striking feature of this condition, angiographically, is the splaying of the carotid bifurcation in the lateral projection (sometimes also seen in the anteroposterior projection) with the internal carotid artery pushed laterally and posteriorly and the external carotid artery displaced anteriorly and either laterally or medially (Fig. 1). This is followed by the appearance of a large network of tortuous vessels, fine in caliber, which course in a meandering fashion in a comparatively well demarcated area in relation to the carotid bifurcation (Fig. 2 and 3). An intense "flush" or "pooling" phase follows which drains slowly into large dilated tortuous veins (Fig. 4). The blood supply is seen to be derived almost entirely from large branches of the external carotid artery but some branches from the thyrocervical trunk of the subclavian artery also contribute to this vascu-

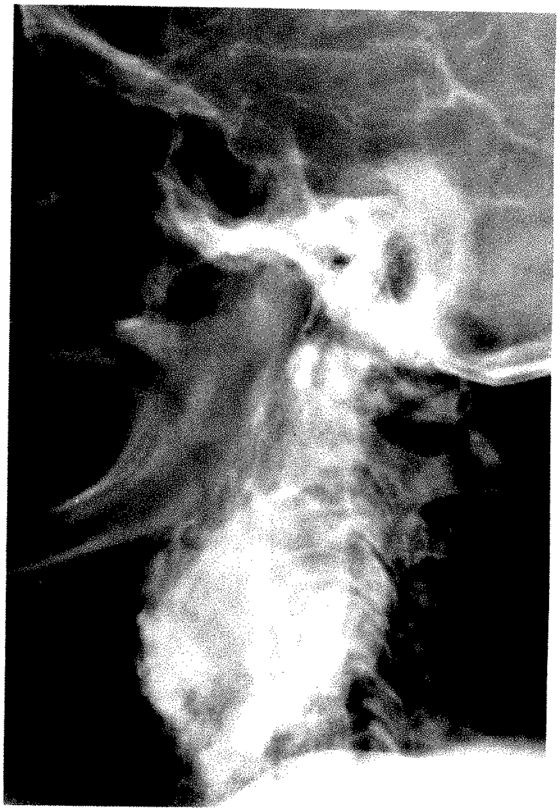

FIG. 4. Angiogram in the venous phase demonstrates pooling of contrast medium in the huge carotid body tumors with beginning of venous drainage noted in dilated veins posteriorly.

lar tumor as was seen in the present case.

More than one-half of the patients whose angiograms are shown in the literature demonstrated the typical features described above.[1,3–5,7,9,13,15–18] A few cases showed displacement of the external and internal carotid arteries in other directions (mainly anterior or lateral displacement of the internal carotid artery), but the tumor in all instances was in intimate relationship to the bifurcation and the typical vascular pattern was seen.[4] There was no reported case in the literature where angiography was performed and these typical features were not present.

Over twenty cases of bilateral carotid body tumors have been described.[1] Two cases had bilateral angiograms that showed both lesions.[1,3] In 1 of these, the angiogram demonstrated an unsuspected small carotid body tumor on the opposite side, in addition to the clinically suspected lesion.[3] As far as we can determine, the present case is the first in which the tumors were investigated using the aortic arch approach and in which the bilateral carotid body tumors were visualized simultaneously.

The surgical approach to the treatment of these tumors depends to a great extent on the angiographic appearance of the lesion. If the tumor is small and does not surround or encase the carotid vessels, then elective surgical excision is justified and is generally without hazard. On the other hand, if the tumor is large and intimately involves the carotid arteries, then surgical intervention, including biopsy, is not indicated since the surgical treatment of this type of lesion has been associated with a high mortality and morbidity.[12] In most instances, the tumor under these conditions cannot be removed without sacrifice of the carotid arteries, a maneuver resulting in a 40 to 50 per cent mortality.[6,8] Occasionally, there is difficulty in controlling bleeding following biopsy of these tumors.[10,12] It is felt that the angiographic appearance is sufficiently characteristic to negate the need for histologic verification.

Surgical intervention might be necessary if the tumor is rapidly growing (suggesting the rarely occurring malignant degeneration of these tumors) with obstruction to carotid flow or resulting pressure on the pharynx. In these rare cases if carotid resection is performed, an attempt should be made to bridge the defect with a vascular graft. Before a resection of the carotid artery is contemplated, carotid arteriography to evaluate possible crossover cerebral circulation should be performed.

In those instances in which surgery cannot be performed, a trial of radiation therapy should be considered. In the bulk of the cases reported in the literature treatment with this modality was disappointing, but there are a few cases described which responded well.[16] In our patient, response to radiation therapy was disappointing.

SUMMARY

A case of a bilateral carotid body tumor is presented along with the roentgenographic findings. The angiographic features are considered to be specific for this diagnosis, can determine the course of therapy, and will enable the surgeon to avoid a potentially hazardous biopsy in nonresectable cases. The use of angiography, in particular the aortic arch approach as performed in this case, in the study of atypical masses in the neck is demonstrated.

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ABERRANT RIGHT SUBCLAVIAN ARTERY

DISPUTE OF COMMONLY ACCEPTED CONCEPTS

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WHEN the right subclavian artery arises as the last branch of a left aortic arch instead of from the innominate artery, the term aberrant right subclavian artery is proper (Fig. 1).

In several large necropsy series the incidence of this anomaly is consistently reported as approximately 1 in every 200 persons.[1,14,37,38]

A major purpose of this paper is to dispute two commonly accepted concepts pertaining to this incomplete vascular ring: first, the frequently quoted figures given for the pretracheal or preesophageal course of this anomalous vessel, and second the cause and effect relationship of aberrant right subclavian artery and "dysphagia lusoria."

This paper is based on a review of a large number of necropsy cases and of cases in which aberrant right subclavian artery was diagnosed roentgenologically; these cases were encountered at this institution.

EMBRYOLOGY

The development of the normal aortic arch system and the formation of an aberrant right subclavian artery have been dealt with comprehensively by Barry[5] and Congdon,[16] and, therefore, will be presented only briefly.

In the young embryo, blood is pumped from the heart into the primitive truncus arteriosus, which ends in a dilatation called the aortic sac. The six-paired aortic arches diverge laterally and posteriorly from this sac to connect with paired vessels called the dorsal aortic roots. The aortic roots are fused in the midline to join the dorsal aorta. Intersegmental arteries that supply the adjacent somites arise from the dorsal aortic roots and the dorsal aorta; these have been numbered correspondingly in Figure 2. The

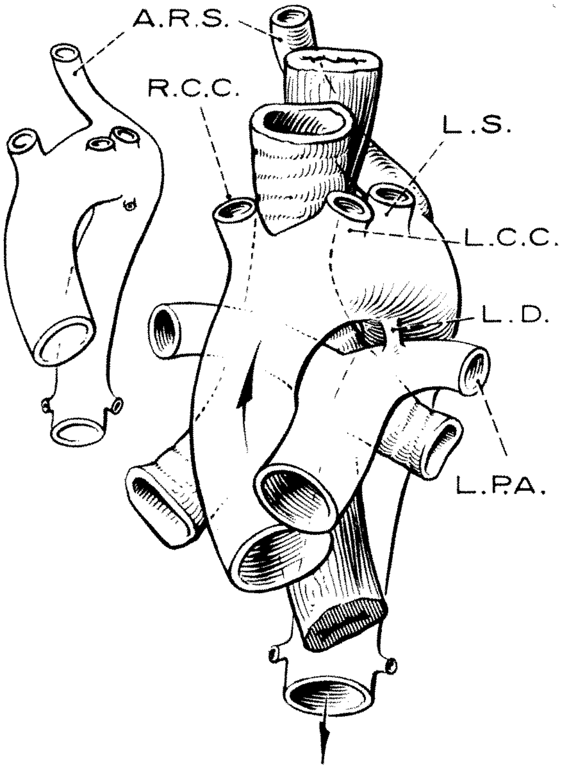

Fig. 1. Diagram showing aberrant right subclavian artery (as viewed from the front) arising as the fourth branch of the aortic arch. *Upper left* shows the anomaly, with trachea and esophagus not shown and *main illustration* shows the relationship of aorta and anomalous vessel to trachea and esophagus.

Abbreviations (also used in Figure 7, *a–c*) are as follows: A.A. = ascending aorta; A.R.S. = aberrant right subclavian artery; D.A. = descending aorta; E. = esophagus; L.C.C. = left common carotid artery; L.D. or L.A. = left ductus arteriosus or ligamentum arteriosum; L.P.A. = left pulmonary artery; L.S. = left subclavian artery; P.A. = pulmonary artery; R.C.C. = right common carotid artery; and T. = trachea. (From Stewart, J. R., Kincaid, O. W., and Edwards, J. E. An Atlas of Vascular Rings and Related Malformations of the Aortic Arch System. Charles C Thomas, Publisher, Springfield, Illinois, 1964, 171 pp.)

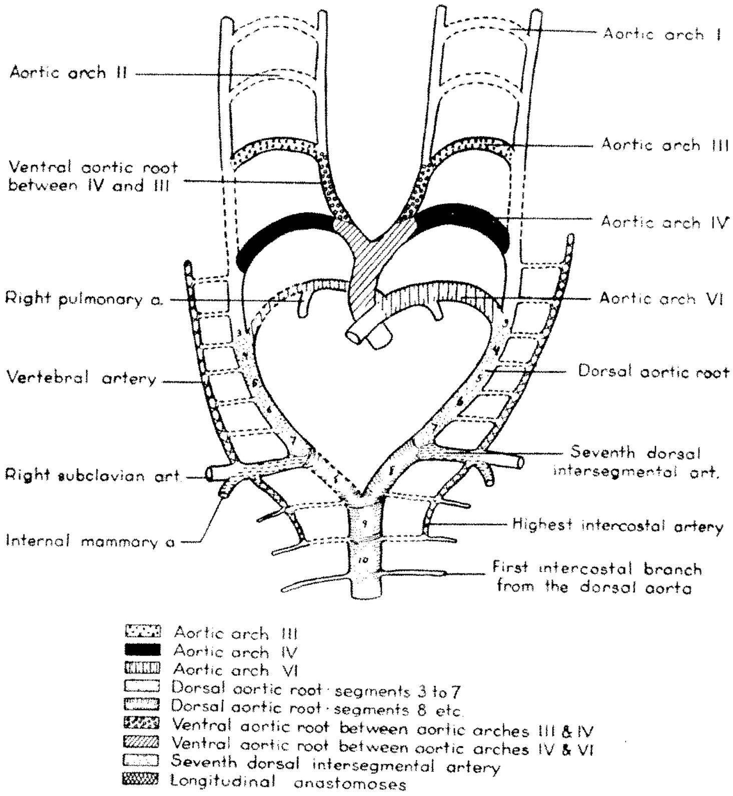
Aortic arch I
Aortic arch II
Aortic arch III
Ventral aortic root between IV and III
Aortic arch IV
Right pulmonary a.
Aortic arch VI
Vertebral artery
Dorsal aortic root
Right subclavian art.
Seventh dorsal intersegmental art.
Internal mammary a.
Highest intercostal artery
First intercostal branch from the dorsal aorta
Aortic arch III
Aortic arch IV
Aortic arch VI
Dorsal aortic root · segments 3 to 7
Dorsal aortic root · segments 8 etc.
Ventral aortic root between aortic arches III & IV
Ventral aortic root between aortic arches IV & VI
Seventh dorsal intersegmental artery
Longitudinal anastomoses

Fig. 2. Schematic diagram indicating various components of the human embryonic aortic arch complex. Those components that do not normally persist in the adult are indicated by broken lines. The paired seventh intersegmental arteries enlarge to form the subclavian arteries. (From Barry, A. The aortic arch derivatives in the human adult. *Anat. Rec.*, 1951, *111*, 221–238.)

first, second, and fifth arches disappear. The third arches are involved in the formation of the common carotid artery complex. Of the six-paired aortic arches, the fourth and sixth are of prime concern in the development of the aortic arch system.

With the regression of the eighth segment of the right dorsal aortic root and the cephalad migration of the subclavian arteries, the left fourth aortic arch enlarges and the final anatomic configuration begins to unfold (Fig. 3 and 4).

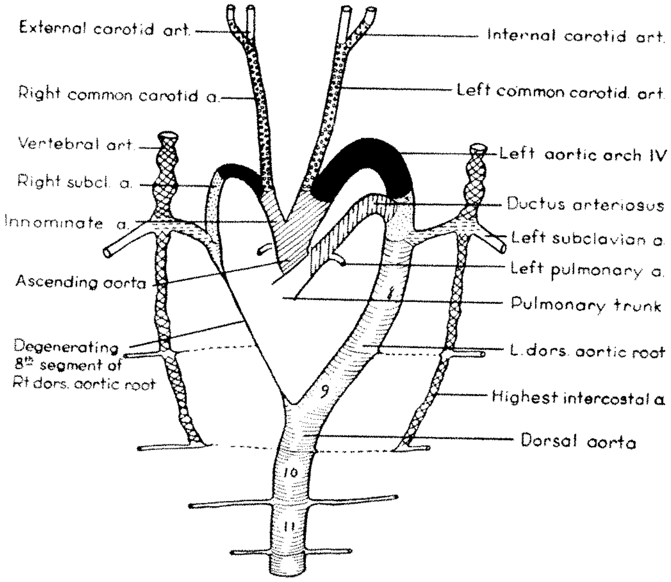

Fig. 3. Diagram of ventral view of aortic arch complex as it appears in a human embryo of 15 mm. crown-rump length. Of key significance in the formation of normal aortic arch is regression of the eighth segment of the right dorsal aortic root. With this occurrence the right subclavian artery has a common origin with the right common carotid artery. Various components are indicated by same scheme of symbols as was used in Figure 2. (From Barry, A. The aortic arch derivatives in the human adult. *Anat. Rec.*, 1951, *111*, 221–238).

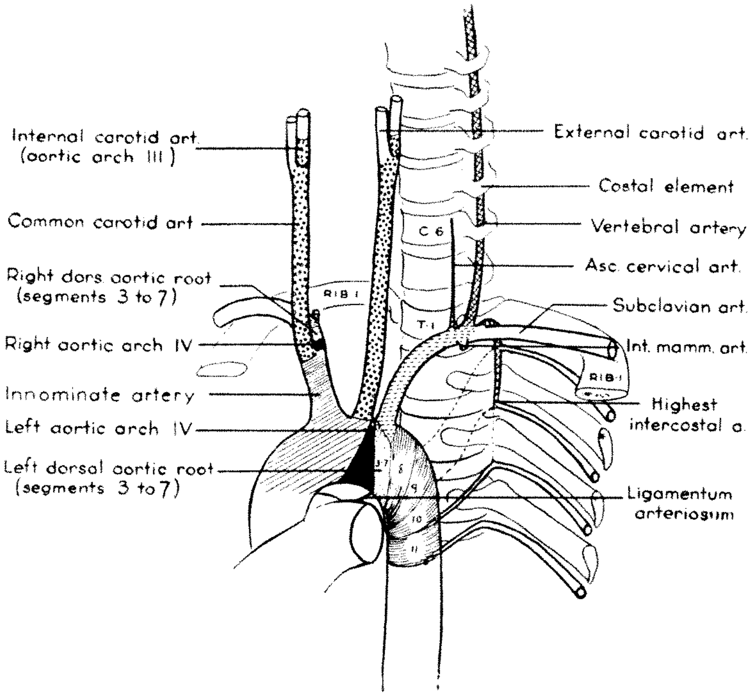

Fig. 4. Diagram of ventral view of resultant normal aortic arch. (Same scheme of symbols as was used in Figure 2.)

In the formation of an aberrant right subclavian artery, regression takes place in the right fourth aortic arch rather than in the eighth segment of the right dorsal aortic root (Fig. 5). Thus, the interruption takes place proximal to the origin of the right subclavian artery which, therefore, arises as the last branch of the aortic arch complex. With the cephalad migration of the subclavian arteries, the final configuration of an aberrant right subclavian artery is present (Fig. 1 and 6).

MATERIAL

The 106 cases in this study are divided into two categories: proved and unproved.

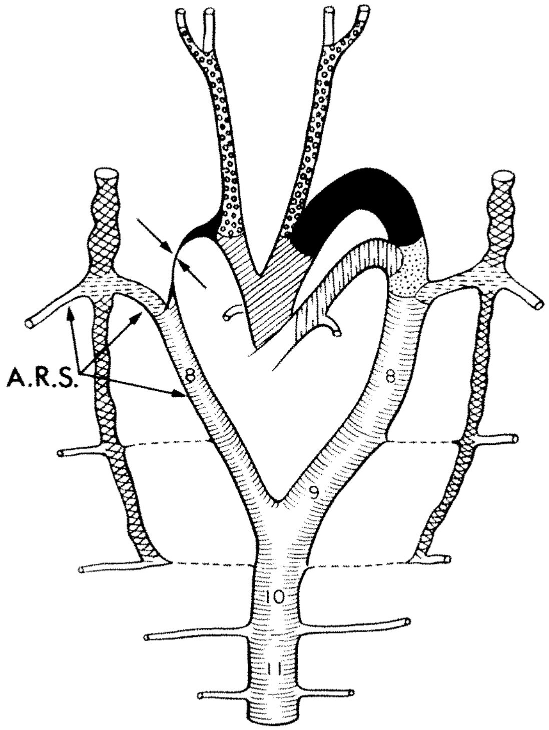

Fig. 5. Diagram of ventral view of embryonic aortic arch complex as it appears when an aberrant right subclavian artery develops. With Figure 2 as a reference, note that instead of the eighth segment of the right dorsal aortic root regressing as in the normal, regression occurs in the right fourth arch (un-lettered arrows). As a result, an aberrant right subclavian artery is formed from the eighth segment of the right dorsal aortic root and its continuation, the right seventh intersegmental artery. Formation of the remainder of the aortic arch is otherwise like that of the normal. (Illustration designed using Barry's scheme of representation.)

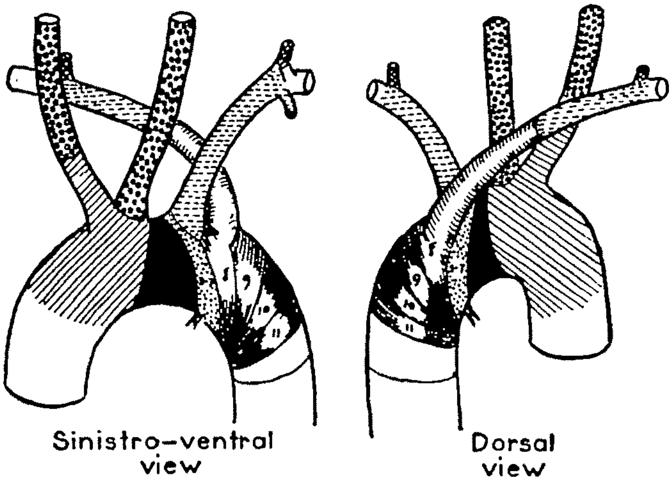

Fig. 6. Diagram of ventral and dorsal views of an aberrant right subclavian artery arising as the fourth branch of the aortic arch.

The proved series comprises 42 cases and includes all available necropsy cases in which an aberrant right subclavian artery was encountered (Fig. 7, *a*, *b* and *c*). The unproved series comprises 64 cases and includes those diagnosed roentgenographically during the years 1953 through 1962 but which were not confirmed at operation or necropsy.

FINDINGS

Of the 106 cases the anomaly occurred in 76 females and 30 males. The youngest patient was a stillborn infant, and the oldest was 89 years of age.

In the 106 cases of aberrant right subclavian artery, dysphagia of some degree was present in only 5 instances. In none of these 5 cases was the dysphagia thought to be secondary to the presence of the aberrant right subclavian artery.

NECROPSY

In each of the necropsy cases and in every case that was diagnosed roentgenographically, the aberrant right subclavian artery was found to pass posterior to both the trachea and the esophagus in its course to the right arm.

In 18 of the 42 necropsy cases, the aberrant right subclavian artery was associated with the presence of other congenital malformations of the heart and great vessels, and in the remainder the aberrant artery occurred as an isolated anomaly.

ROENTGENOGRAPHY

The roentgenographic features of an aberrant right subclavian artery were first described by Kommerell[27] in 1936. It is our impression that the aberrant vessel may be present without causing deformity of the esophagus and, therefore, may not be recognizable in a study of a barium-filled esophagus. However, if sufficient compression is present, as it was in all 64 cases in our series, the location of the defect and the defect itself are consistent and characteristic.

In the anteroposterior view, a shallow groove is seen in the barium-filled esophagus at and slightly above the level of the aortic knob (Fig. 8*a*). This groove angles upward from left to right corresponding to the course of the vessel toward the superior aspect of the right first rib. The oblique projections of the barium-filled esophagus may demonstrate the esophageal compression extremely well (Fig. 8*b*). The left anterior oblique projection often is deceiving, since the appearance of the esophagus may suggest a preesophageal course of the aberrant right subclavian artery, whereas a true lateral projection clearly demonstrates the vessel posterior to the esophagus. In the lateral projection of the barium-filled esophagus, the aberrant right subclavian artery produces a smooth, shallow defect in the posterior wall of the esophagus (Fig. 8*c*). This concave defect is much smaller than the posterior compression defect produced by a double aortic arch and other complete vascular rings.

A sometimes confusing esophageal impression is that produced by the left main bronchus as it passes to the hilus of the left lung. This indentation is frequently seen on roentgenograms of the barium-filled esophagus and can be distinguished from an aberrant right subclavian artery since the indentation produced by the bronchus lies below the aortic knob and is seen in the anterior wall of the esophagus when viewed in the lateral projection.

DISCUSSION

COURSE OF THE ABERRANT RIGHT SUBCLAVIAN ARTERY

Since the original description of an aberrant right subclavian artery by Hunauld[25] in 1735, many reports of this anomaly have appeared in the medical literature.[3,10,14,24,34,37,38] In the reports in the current literature it is usually stated that the anomalous right subclavian artery, in its course to the right arm, passes behind the esophagus in 80 per cent, between the trachea and esophagus in 10 to 15 per cent, and in front

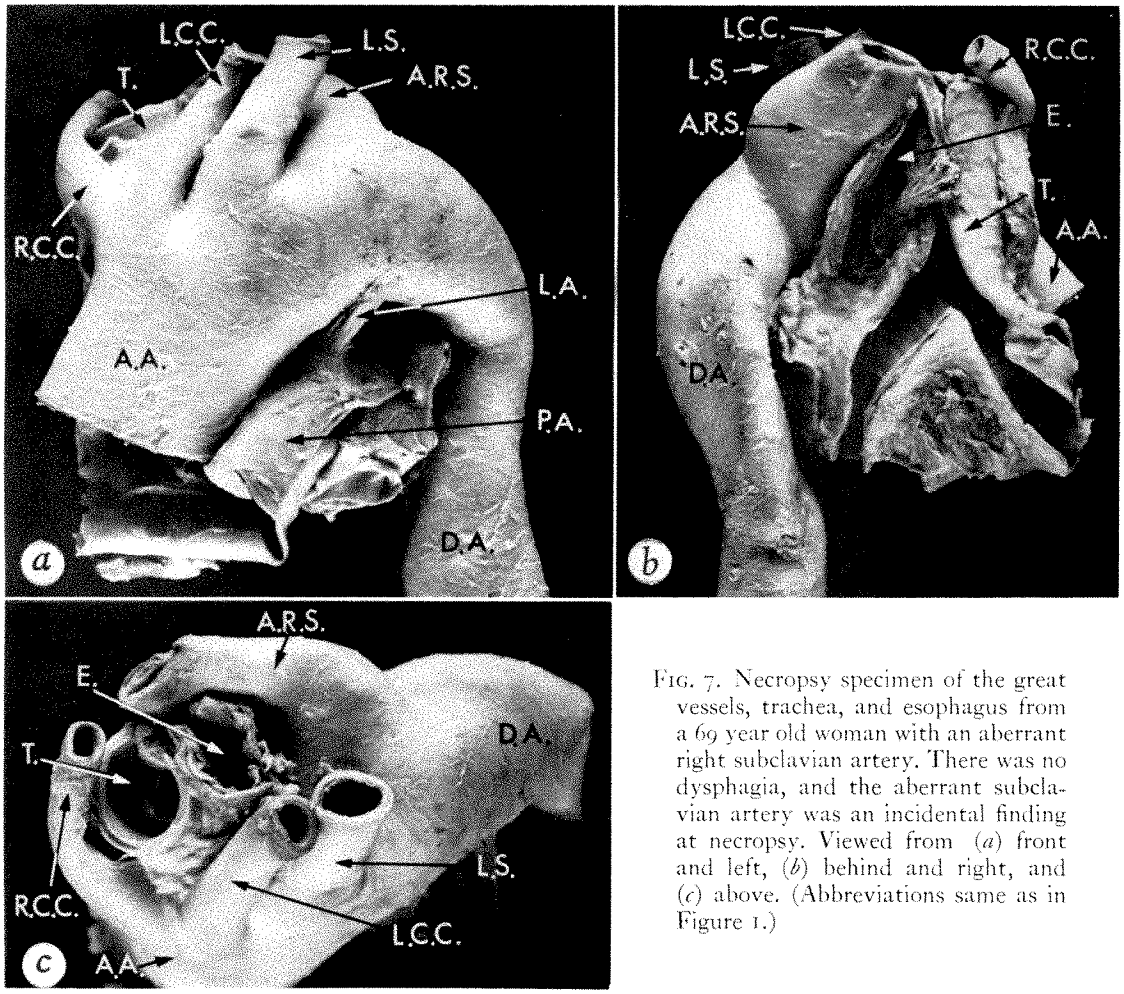

Fig. 7. Necropsy specimen of the great vessels, trachea, and esophagus from a 69 year old woman with an aberrant right subclavian artery. There was no dysphagia, and the aberrant subclavian artery was an incidental finding at necropsy. Viewed from (*a*) front and left, (*b*) behind and right, and (*c*) above. (Abbreviations same as in Figure 1.)

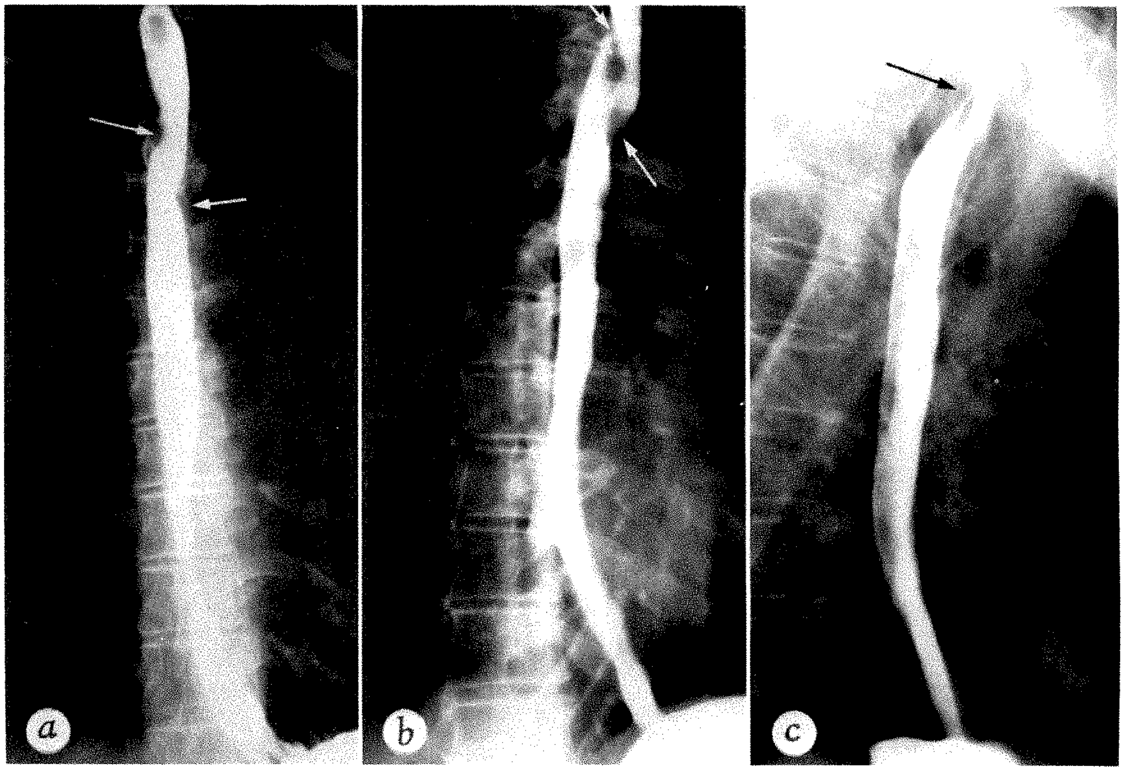

FIG. 8. Roentgenographic views of barium-filled esophagus as observed in a 50 year old woman. No history of dysphagia was given. Compression of esophagus (arrows) is typical of aberrant right subclavian artery. (*a*) Anteroposterior, (*b*) right anterior oblique, and (*c*) left lateral views.

of the trachea in 5 to 10 per cent of the cases.[2,7,13,18,39,46] After a reasonably comprehensive review of the literature, we observed that, almost without exception, the current authors who quoted these figures have never personally encountered a case proved at necropsy in which the anomalous vessel passed either in front of the trachea or between the trachea and the esophagus. If any references were given in support of these figures, they invariably and eventually led back to an article by Holzapfel[24] in 1899.

Holzapfel, in a search of the literature up to 1899, found 133 cases (including 5 of his own, each of which had a retroesophageal aberrant right subclavian artery) in which he believed that there was sufficient anatomic description to warrant inclusion in his review. Of the 133 cases, 20 cases (15 per cent) were presented as examples in which the aberrant subclavian artery passed between the trachea and the esophagus and 6 cases (5 per cent) were presented as examples in which the anomalous vessel passed in front of the trachea. In each of our necropsy specimens of aberrant right subclavian artery and in every example we could find in the current literature in which a necropsy specimen was specifically reported, the anomalous vessel coursed behind the esophagus to reach the right arm. Therefore, it seemed advisable to search out and analyze critically the references included by Holzapfel as examples in which the anomalous vessel passed in front of the trachea or between the trachea and the esophagus.

Of the 26 cases cited by Holzapfel as examples of pretracheal or preesophageal courses for an aberrant right subclavian artery, 24 were available for review. In 6 of these, included by Holzapfel as examples of an aberrant right subclavian artery passing between the trachea and the esophagus, the original authors failed to mention the relationship of the right subclavian artery to the esophagus.[12,18,25,28,30,46] In 5 other cases the original authors did not report their personal observations but either described observations of others or discussed the anatomy of the anomaly in general terms.[8,11,20,33] Descriptions in 2 cases are so indefinite that it is impossible to tell what anomaly they represent.[17,35] An additional case is based only on a verbal communication between the original observer and Mears.[29] Two cases, both described by Walter,[44] indicate that the anomalous right subclavian artery passed anterior to the trachea. The drawing of one of these cases does not correspond to the author's description of the specimen, and the trachea and esophagus are not included in either drawing. While Walter's descriptions are clear, it appears improbable that a single author, encountering 2 specimens of aberrant right subclavian artery, should find both to have a pretracheal course.[38] Two of the original articles could not be obtained for review.[23,42]

Of the 24 cases available for review, only 8 case reports were found in which it was clearly stated that the aberrant vessel passed between the trachea and the esophagus or in front of the trachea.[4,6,9,22,26,31,36,40] In addition, 2 other cases, not cited by Holzapfel, were reported in the nineteenth century in which the authors definitely stated that an anomalous right subclavian artery passed between the trachea and the esophagus.[15,43] Since the reports of these 2 cases prior to 1890,[15] the only subsequent report that could be found describing such a course of the anomalous vessel is that of Scheldrup.[39] In 1957, Scheldrup reported that an anomalous right subclavian artery may pass between the trachea and the esophagus or in front of the trachea and stated that "twenty cases of this latter position of the artery have been reported by Holzapfel, and the author recently dissected an additional specimen of this anomaly in an adult cadaver. These 21 case reports make untenable the unsubstantiated premise that the anomalous right subclavian artery never passes between the trachea and esophagus."

Notwithstanding the care and effort of medical writers, inadvertent errors of observation are occasionally made and appear in the literature. This was especially true in the eighteenth and nineteenth centuries—in fact, a few authors of that era stated that the "usual" course of the anomalous right subclavian artery, on its way to the right arm, is between the trachea and the esophagus.[33] Although it is obviously impossible to deny that such variations have been reported, they are very difficult to explain embryologically.

The present authors feel that cases of an aberrant right subclavian artery passing in front of the trachea or between the trachea and the esophagus, if they exist at all, are extremely rare.

RELATIONSHIP OF DYSPHAGIA AND ABERRANT RIGHT SUBCLAVIAN ARTERY

The concept that an aberrant right subclavian artery produces dysphagia originated in 1794 when Bayford[6] colorfully described the clinical history and death of one Jane Fordham. Her death was attributed to progressive dysphagia and starvation. The only finding at necropsy that was considered significant was an aberrant right subclavian artery. From this experience, Bayford coined the term "dysphagia lusoria" which translated means "difficulty in swallowing due to a trick of nature." Although Bayford's cause and effect relationship between the anomalous right subclavian artery and dysphagia was not well accepted at that time,[21,26,38] the term remained popular. Interest was re-established in 1946 when Gross[19] initially employed operative division of the vessel in an infant who presented as a feeding problem.

Applying statistics of the incidence of this anomaly from studies at necropsy to the population of the United States today there should be at least 900,000 people with aberrant right subclavian artery. It should also be remembered that dysphagia and feeding problems are not uncommon and are frequently without apparent cause. It is only logical, therefore, to occasionally find an infant presenting as a feeding problem or an adult with dysphagia in whom an aberrant right subclavian artery is present. To ascribe these symptoms to compression of the esophagus by an aberrant right subclavian artery should, therefore, be subject to careful scrutiny—*post hoc ergo propter hoc*. Should these symptoms be temporary in a given patient, or should there be a psychogenic component on the part of the mother whose child is a feeding problem or on the part of the adult with dysphagia, a thoracotomy in itself might be expected to offer relief by the very nature of this dramatic and time-elapsing mode of treatment.

From a physiologic point of view, there is no dilatation of the esophagus above the site of an anomalous right subclavian artery, nor is there any significant obstruction to the flow of barium through this portion of the esophagus. The aortic knob in the elderly person often impinges on the esophagus with more pressure than an aberrant right subclavian artery, the latter pressure defect typically being shallow. Certainly, if the aberrant vessel becomes aneurysmal or if other vascular anomalies (such as a right ductus arteriosus) exist, forming a complete vascular ring, dysphagia might be expected.[41]

In reviewing the case histories of the patients who constitute this study, we found an impressive lack of esophageal obstructive symptoms. To our knowledge, no patient has ever been operated on at this institution for relief of dysphagia produced by an aberrant right subclavian artery. Recalling Bayford's article, we cannot help but wonder if the long suffering Jane Fordham did not die of another disease, such as scleroderma. While it is impossible to say that an aberrant right subclavian artery never causes significant esophageal obstruction, certainly such a cause and effect relationship has been enormously overrated. In any patient exhibiting dysphagia or feeding difficulty in whom an aberrant right subclavian artery is found, a careful search for a cause other than this vascular anomaly should be made. If no other cause is found, one must still entertain a reasonable degree of doubt as to the actual role this anomaly has in the production of symptoms. In the final analysis, the morbidity and risk of thoracotomy must be carefully weighed against the likelihood that this anomaly is the causative factor and against the degree of disability present.

SUMMARY AND CONCLUSIONS

A series of 106 cases of aberrant right subclavian artery, comprised of 42 cases studied at necropsy and 64 cases diagnosed roentgenologically, is presented.

In this study it was found that, for all practical purposes, an aberrant right subclavian artery always passes behind the esophagus to reach the right arm, although a few reports exist in the literature in which it is stated that the aberrant vessel passed between the trachea and the esophagus or in front of the trachea to reach the right arm. From future detailed case reports perhaps it can be definitely determined whether in rare instances this aberrant vessel passes other than posteriorly to the esophagus.

The role an aberrant right subclavian artery has in the production of dysphagia in adults or feeding problems or other symptoms in infancy is greatly overrated. It is doubtful that this isolated anomaly ever produces significant dysphagia.

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CALCIFICATION IN THE CORONARY ARTERIES AND ITS RELATIONSHIP TO ARTERIOSCLEROSIS AND MYOCARDIAL INFARCTION*

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CALCIFIED lesions in the coronary arteries, long recognized postmortem, deserve some critical assessment of their relationship to clinical manifestations of coronary heart disease. Although the process of calcification is incompletely understood,[11,16] the lesions found in coronary arteries have usually been interpreted as indicators of a late stage of arteriosclerosis.[3,15] Some elucidation of the role of calcification in coronary arteriosclerosis appeared especially appropriate at this time, since recent roentgenologic advances show promise of detecting these lesions. Reference has been made lately to the ease with which small radiopaque flecks,[10] even when in rapid motion, can be detected by cinefluorography. Thus, new information about the condition of the walls of the coronary arteries may become available through future examination procedures.

Surprisingly, there are very few objective data on the relationship of coronary calcification to the amount of coronary arteriosclerosis and to the incidence of myocardial infarct. If either of these relationships were found to exist, particularly the latter, then the detection of calcified lesions by cinefluorography may have practical value in the clinical diagnosis of arteriosclerotic heart disease. Some assessment of these relationships was possible from postmortem data of the pathology and epidemiology of coronary artery disease obtained in a previously described study.[2] In such an assessment these questions are posed: (1) do more severe degrees of coronary arteriosclerosis tend to accompany more extensive calcification in the walls of the coronary arteries and (2) does myocardial infarction tend to occur more frequently with more extensive calcification in the coronary arteries?

MATERIAL AND METHODS

The present material comprises 904 essentially consecutively autopsied individuals who died from any cause and who were 15 to 84 years of age. All had died at Albany Medical Center Hospital, Albany, New York, within the period from January, 1960 to December, 1962.

Examination of the heart was performed in two stages: (1) gross examination in the fresh state, excluding the coronary arteries; and (2) examination of the coronary arteries after 1 week of formalin fixation.

1. The examination of the fresh heart consisted of a detailed examination of its components, with particular emphasis on the myocardium for signs of fresh or healed infarction or of focal fibrosis. Zones of scarring or ischemic necrosis were classified as infarcts if they exceeded 1.0 cm. in size. If infarction, scarring or ischemic necrosis (as defined above) was present, it was charted on a special form.

2. The fixed coronary arteries were examined according to a method previously described in detail.[7] In brief, this method is based on the premise that the amount of coronary arteriosclerosis is reflected, at least in large part, by coronary arterial wall thickness. This premise has recently been validated by a histologic study in which micrometric measurements of diseased portions of coronary arteries have indicated that where wall thickening had occurred it was the direct result of the arteriosclerotic process.[9] As currently practiced, after for-

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malin fixation, regular 5 mm. cuts (using a special marking knife) are made in the main coronary arterial tree until the external diameter of the coronary arteries is only 1.0 mm. Alternate segments of the main left, left anterior descending, left circumflex, and right coronary arteries are removed, and the coronary arterial wall thickness is measured at constant locations in the plane parallel to the epicardial surface, by especially designed calipers. From the multiple measurements made, a mean for each heart is calculated and used as an approximation of the mean coronary wall thickness. At the time the coronary arteries are examined, other findings, such as calcification, thrombosis, old occlusions, hemorrhage into plaques, or ulcerated plaques, are recorded according to their location on a diagrammatic chart. Areas containing calcified substances are recognized by their grittiness when encountered by the cutting knife.

In this report the attempt has been made to assess the process of calcification quantitatively. A subject was categorized according to whether there were significant amounts of calcification in 1, 2, 3, or none of the major branches of the coronary arterial tree. For simplification in the categorization of artery involvement with calcification, those arteries possessing small calcified lesions occupying no more than one quarter of their length were placed in the "no calcification" artery category. Thus, in order for an artery to be categorized as being calcified, a lesion had to be present in two or more quarters of its length. This definition of a lesion is referred to herein as a "significant" lesion and was adopted to avoid undue emphasis upon lesions of minute size. Also, due to its short length, the main left coronary artery, *i.e.*, proximal to the origin of the left descending branch, was considered as part of the left descending branch.

RESULTS

In Table I, the coronary artery wall thickness is given according to the presence of significant lesions in no artery, in 1, 2, or 3 arteries and by age and sex. A definite tendency was present in each age group for the mean of the wall thickness to increase as the number of arteries with calcification increased. Wall thickness increased in a step-wise manner with the number of arteries calcified to a highly significant degree.* The thickness was thus found to be greatest, that is, 0.60 mm. or more, in all groups with calcified lesions in 3 arteries. It should be noted that the wall thickness for men below 65 years differed markedly between the extremes of calcification, *i.e.*, those with no calcified arteries as compared with those with three arteries calcified: means of wall thickness varied from 0.40 mm. to over 0.65 mm. This difference in wall thickness has been found crucial at this particular range of disease since here a marked tendency toward infarction has been found to occur.[8,12]

The distribution of subjects according to the presence or absence of myocardial infarction along with the number of coronary arteries with significant calcification is presented in Table II by age and sex. Opposing patterns of coronary artery calcification are evident in the two major classes, *i.e.*, those with and without an infarct. Subjects without an infarct showed a tendency for fewer arteries to be affected with calcification, while those with an infarct showed the opposite trend, *i.e.*, a tendency for more arteries to be affected. This is especially evident in the younger groups of both sexes. In the oldest groups, males over 64 and females over 74, although the pattern of differences is not as striking as in the younger groups, subjects with an infarct repeatedly showed more arteries affected with calcification than did those without an infarct.

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* Analysis of covariance was applied to subjects aged 45–84, separately for males and females, and using age as a covariable. Highly significant F values were obtained. To judge if coronary artery wall thickness was directly related to the number of arteries with calcification, the following differences (contrasts) among means were tested, for both sexes, by the S-method of multiple comparison:[19]

$$\mu_1 - \mu_0,\ \mu_2 - \mu_1, \text{ and } \mu_3 - \mu_2,$$

where, μ_i denotes the population mean wall thickness for class i, and $i = 0$, 1, 2 or 3 denotes the number of arteries with calcification. All contrasts except $\mu_2 - \mu_1$ for females were significantly different from zero at $p < 0.01$. The latter was significant at $p < 0.10$.

Table I

CORONARY ARTERY WALL THICKNESS (CAWT), ACCORDING TO THE NUMBER OF CORONARY ARTERIES WITH CALCIFICATION AND BY AGE AND SEX IN 904 AUTOPSIED SUBJECTS, ALBANY MEDICAL CENTER, 1960–1962

Age and Sex	No. of Arteries with Calcified Lesions*							
	None		One		Two		Three	
	Mean CAWT (mm.)	No. of Subjects	Mean CAWT (mm.)	No. of Subjects	Mean CAWT (mm.)	No. of Subjects	Mean CAWT (mm.)	No. of Subjects
Males								
All ages	.39	141	.47	90	.53	132	.64	146
15–44	.35	48	.43	5	.44	5	.72	4
45–54	.41	30	.44	16	.53	15	.68	13
55–64	.39	34	.47	27	.53	49	.66	37
65–74	.45	22	.49	23	.52	38	.61	57
75–84	.46	7	.47	19	.56	25	.62	35
Females								
All ages	.34	198	.47	56	.51	75	.60	66
15–44	.31	53	.58	3	.72	1	—	0
45–54	.33	54	.46	8	.45	5	.60	2
55–64	.36	50	.45	13	.48	24	.61	10
65–74	.37	34	.46	19	.49	24	.60	28
75–84	.41	7	.46	13	.56	21	.61	26

* Calcified lesions were defined as those involving more than one quarter of a coronary artery. Study subjects were classified according to the absence of such lesions or their presence in 1, 2, or 3 coronary arteries.

The expected trend of calcified lesions to occur more frequently with increasing age is displayed in Table II in the data for subjects without an infarct. A tendency is evident for males to have calcified lesions more frequently and at an earlier age than females. Among individuals with myocardial infarction, however, an age trend in calcification was not present for males but was suggested for females.

Another view of the relationship between calcification and infarction is presented in Figure 1. The percentage of individuals with infarct is shown for groupings based upon the number of arteries with significant calcified lesions. Subgroupings for age and sex are retained. A significant trend* was found where the percentage of subjects with infarct tended to increase in a stepwise manner as calcification occurred in an increasing number of arteries. The data for females aged 45–54 are presented in only two categories: no artery and 1 or more arteries with calcified lesions. This was done because of the small numbers in the 2- and 3-artery categories of calcification. A significant difference between these two latter groups ($p < 0.001$, chi-square) was found in the occurrence of infarct.

DISCUSSION

The findings presented herein provide evidence for interrelationships among coronary artery calcification, coronary artery wall thickness, and myocardial infarction as these are seen postmortem. Employing a gross quantitation of calcification in the coronary arteries, evidence existed for a direct relationship between the amount of calcification and the amount of arteriosclerosis, as the latter was determined by

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* A significant linear relationship was found between the number of arteries involved with calcification and the percentage with infarct ($p < 0.01$, regression chi-square)[6] in all age groups and in both sexes. For two groups, males aged 45–54 and females aged 55–64, because of small expectations, "number of arteries with calcification" was treated as a three-category variable.

Table II

MYOCARDIAL INFARCT AND CALCIFICATION ACCORDING TO THE NUMBER OF CORONARY ARTERIES WITH CALCIFIED LESIONS, AND ACCORDING TO AGE AND SEX, IN 904 AUTOPSIED SUBJECTS, ALBANY MEDICAL CENTER, 1960–1962

Age and Sex	Subjects without Myocardial Infarct					Subjects with Myocardial Infarct				
	No. of Subjects	Per Cent with Calcification* in:				No. of Subjects	Per Cent with Calcification* in:			
		0 artery	1 artery	2 arteries	3 arteries		0 artery	1 artery	2 arteries	3 arteries
Males										
All ages	349	37.0	20.1	24.9	18.0	160	7.5	12.5	28.1	51.9
15–44	57	80.7	7.0	7.0	5.3	5	40.0	20.0	20.0	20.0
45–54	55	52.7	21.8	21.8	3.6	19	5.3	21.0	15.8	57.9
55–64	95	31.6	20.0	35.8	12.6	52	7.7	15.4	28.8	48.1
65–74	87	20.7	21.8	25.3	32.2	53	7.5	7.5	30.2	54.7
75–84	55	10.9	29.1	27.3	32.7	31	3.2	9.7	32.2	54.8
Females										
All ages	311	61.1	13.2	16.4	9.3	84	9.5	17.9	28.6	44.0
15–44	54	96.3	3.7	0.0	0.0	3	33.3	33.3	33.3	0.0
45–54	62	85.5	6.4	6.4	1.6	7	14.3	57.1	14.3	14.3
55–64	82	58.5	11.0	23.2	7.3	15	13.3	26.7	33.3	26.7
65–74	75	41.3	20.0	21.3	17.3	30	10.0	13.3	26.7	50.0
75–84	38	15.8	28.9	31.6	23.7	29	3.4	6.9	31.0	58.6

* Calcified lesions were defined as those involving more than one quarter of a coronary artery. Study subjects were classified according to the absence of such lesions or their presence in 1, 2, or 3 coronary arteries.

coronary artery wall thickness. The calcification process in the coronary arteries was found related also to the occurrence rate of myocardial infarction. These relationships were found to exist apart from the familiar pattern of an increased frequency of calcified lesions with aging.

These results may prove useful, it is expected, in explorations of cinefluorographic detection of calcification in the coronary arteries. Since individuals with calcified lesions in more than 1 artery were found to be increasingly more likely to have a myocardial infarction, the possibility arises that multiple sites of calcification—and in several arteries—are more readily visible cinefluorographically. These lesions can thus serve as an indicator of more severe degrees of arteriosclerosis and as an alarm signal for future development of myocardial infarction. On the assumption that calcification generally precedes myocardial infarction, patients with certain patterns of coronary calcification may prove to be particularly prone to show a future infarction. If found to be true and if cinefluorography is shown to be useful in detection of calcification, then the procedure may become an excellent method for identifying preclinically affected individuals at high risk for myocardial infarction.

Many aspects of the basic relationship of calcification to arteriosclerosis in the coronary arteries are being studied by others. Evidence for the fact that most gritty parts of the coronary arteries contain substances that are, indeed, opaque to roentgen rays has been presented by Blankenhorn[3] and Blankenhorn and Stern.[5] The latter have demonstrated through roentgenographic examination of the isolated heart postmortem, that these lesions can be detected as small as 3 mm. in their greatest dimension. Evidence for such lesions to contain calcium

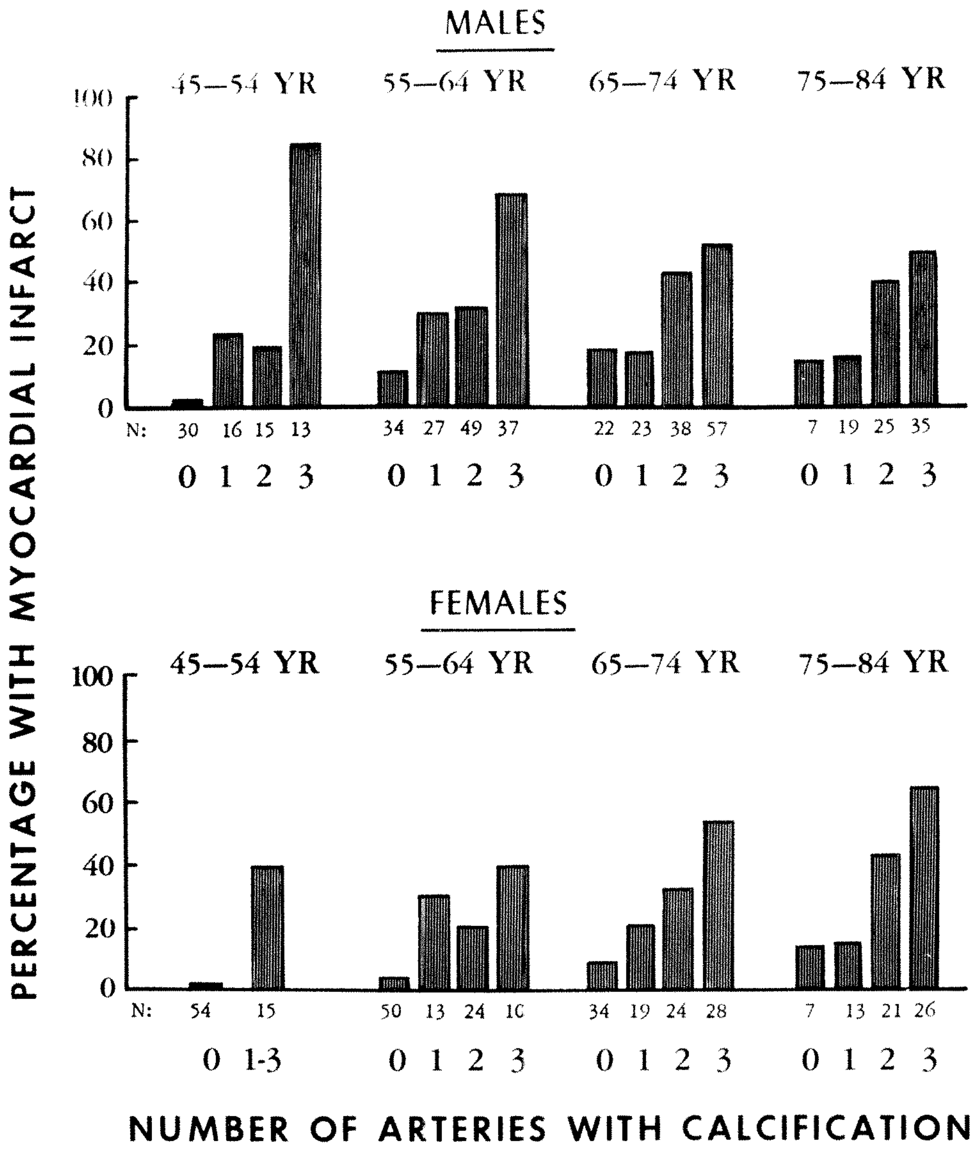

Fig. 1. Percentage of subjects with myocardial infarction, according to number of coronary arteries with calcification and by age and sex. Calcified lesions were defined as described in footnote of Tables I and II. The number (N) below each column refers to the total number of subjects, *i.e.*, those with and without infarct, on which the percentage is based. The data for females aged 45–54 are limited by the small numbers in the 2- and 3-artery categories. Data derived from 904 autopsied subjects, Albany Medical Center, 1960–1962.

has been provided by several investigators.[4,20] Although calcium has been found present in appreciable amounts in most coronary artery specimens,[20] it is the focal concentration, which can be expected to be radiopaque, that is important in the present context. Evidence substantiating the usefulness of cinefluorography in the detection of coronary calcification has been recently described by Jorgens *et al.*[13,14]

The procedures employed in this study possess certain inherent limitations which affect sensitivity in detecting calcification. A portion of calcified lesions must have been overlooked due to size or location. It will be recalled that examinations were made at the site of cross-sectional cuts, 5 mm. apart, thus permitting some smaller lesions to escape detection. A sensitivity level of 62 per cent was found in these data as presented. It will be recalled that small lesions existing in but one quarter of an artery were set aside, however, and grouped with the "no calcification" artery category. When these latter are included with the positives, sensitivity becomes 80 per cent. This level of sensitivity is the same, moreover, as that found by Blankenhorn and Stern[5] in their postmortem roentgenographic studies of the coronary arteries of isolated hearts. The agreement in sensitivity level of the two procedures is probably not quite as close as these results indicate, since the age-sex composition of the two study populations was not entirely comparable. Finally, in regard to sensitivity, mention should be made of a low frequency of calcification in the coronary arteries, *i.e.* 7 per cent, which has been reported by Morris[17] from a National Necropsy Survey in England. Other findings derived from autopsied subjects in England[18] suggest, however, that calcification can be found with severalfold greater frequency.

The possibility exists, of course, in autopsy data that factors causing selection have affected in subtle ways relationships such as these described in this report. Specifically, in the context of the present study, individuals with myocardial infarction who tended to have more coronary calcification through some selective factors may have been brought into the study group. Influences possibly bringing about such kinds of selection cannot be readily envisioned without more knowledge of the physiology and pathology of calcification. One aspect of selection (not shown) was examined briefly. Data pertaining to individuals studied earlier were divided into two groups and compared: those certified (on death certificates) as arteriosclerotic heart disease and those certified as other causes. Both groups showed an association, within age-sex subgroups, between artery calcification and myocardial infarct which was similar to that described in this report. It should be added that the group certified as arteriosclerotic heart disease does not by any means include all cases with myocardial infarct but, instead, only about one half of such cases can be expected to fall into the group.[1] It seems most reasonable to conclude on the basis of present knowledge that the relationships found herein between infarction and calcification, and the amount of coronary arteriosclerosis and calcification, are more plausible as biologic relationships than as associations resulting from selection.

SUMMARY

Necropsy data pertaining to myocardial infarction, arteriosclerosis, and calcification of the coronary arteries have revealed interrelationships in their occurrence. As calcification was found in more coronary vessels, myocardial infarction and greater levels of coronary arteriosclerosis appeared increasingly more probable. It is anticipated that these findings will be useful in explorations of coronary calcification by cinefluorography.

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CORONARY ARTERIOGRAPHY*

SOME OBSERVATIONS ON TECHNIQUE AND INTERPRETATION

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CORONARY angiography has recently gained popularity due to the work of Sones[16] and others, and because of increasing interest in investigation of coronary artery disease. The different techniques that have been used are summarized in Table I.

Precise selective injection of contrast medium into a single artery (selective technique) is the method of choice for optimum visualization of any vascular territory and this method of arteriography provides the maximum visualization of the coronary arteries in humans. Sones and Shirey[15] and Ricketts and Abrams[13] have described special catheters for selective cannulation of the coronary arteries in order to accomplish this result.

The following observations have been obtained from experience with animal experiments over a 3 year period and clinical studies of 70 patients.

TECHNICAL CONSIDERATIONS

(A) CATHETER DESIGN

None of the present commercially available catheters fulfill the characteristics of an ideal design. These requisites are a soft, flexible, tapered, radiopaque tip and a relatively semirigid body of inner material which will permit adequate maneuverability in all planes. The tip should have a variable range of motion, be small enough to be easily introduced and safely left in position for multiple injections and be firm enough for percutaneous introduction. A catheter which closely resembles these characteristics has been designed in our laboratory (Fig. 1). This catheter has a double curve. The distal one is under the control of a metallic deflector which straightens the tip when advanced and allows it to curve when retracted. The proximal curve stabilizes the catheter by banking off the wall opposite the cannulated orifice. There are two outlets at the proximal end of the catheter. One permits continuous monitoring of pressure and infusion of heparinized glucose solution. The other outlet is used for the injection of radiopaque contrast material.

(B) METHOD FOR INJECTION OF CONTRAST MATERIAL

Using the Sones catheter and hand injection of contrast medium (2 to 6 cc. of warmed 75 per cent hypaque*), some of our cineangiographic studies have demonstrated thread-like coronary arteries which we initially interpreted as "abnormally narrowed by atherosclerosis." Because of difficulties in interpretation encountered when hand injecting through the small-tip Sones catheter, we decided to use a pressure injector. A total volume of 4 to 6 cc. of 75 per cent hypaque was injected under pressure in a short period of time. It was surprising to see that vessels which appeared "diffusely narrowed" when hand-injected were adequately filled and normal in appearance in sequences obtained with the pressure injector (Fig. 2, *A* and *B*). After these comparisons, we decided to use pressure injection for all coronary angiographic studies.

(C) RECORDING OF THE IMAGE

Our initial coronary angiographic studies were recorded by biplane cinefluorographic

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TABLE I

HISTORY OF CORONARY ANGIOGRAPHY

Year	Author	Comment
1933	Rousthöi[14]	Demonstrated the coronary arteries in the intact experimental animal
1942	Castellanos *et al.*[4]	Venous angiocardiography in man. Occasional demonstration of the coronary arteries
1952	Di Guglielmo and Guttadauro[5]	First successful method of coronary artery visualization by aortography
1958	Dotter and Frische[6]	Occlusion aortography
1958	Arnulf[2]	Acetylcholine-induced arrest
1959	Lemmon *et al.*[11]	Suprasternal transthoracic coronary arteriography. Acetylcholine-induced arrest
1959	Sones[16]	Selective coronary arteriography
1960	Jönsson and Hellström[9]	Percutaneous femoral retrograde aortography with an Ödman catheter
1960	Nordenstrom[12]	Induced reduction of intrathoracic circulation
1960	Williams *et al.*[19]	Loop-end catheter
1961	Lang and Sabiston[10]	Sinus flush technique
1962	Amplatz and Harner[1]	Percutaneous subclavian retrograde aortography
1962	Ricketts and Abrams[13]	Selective coronary arteriography
1962	Gensini *et al.*[7]	Coronary venography in man
1963	Gensini[8]	New teflon catheter for percutaneous catheterization and contrast material injection

technique. This proved to be cumbersome and technically unsatisfactory. With the advent of a rotating table-top cradle designed in our laboratory, monoplane studies proved to be more satisfactory. This rotating cradle (Fig. 3, *A*, *B* and *C*) is portable and can be adapted to any roentgenographic table. It is motorized, can be controlled remotely and at variable speed permitting rotation of the patient from 0 to 90°

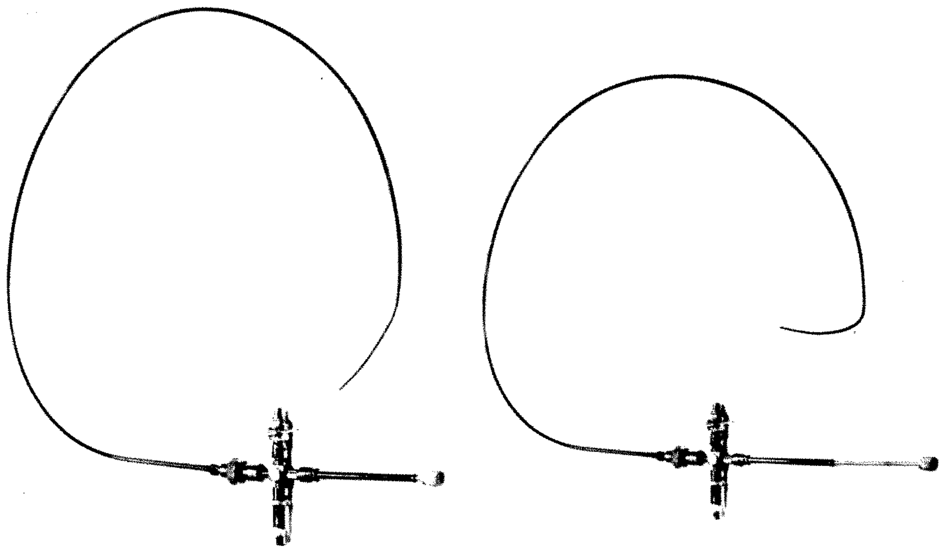

FIG. 1. Specially designed catheter with complete external control of the movement of the tip. *Left.* Metallic deflector in advanced position straightens tip. *Right.* Withdrawal of deflector restores sharp curve to tip.

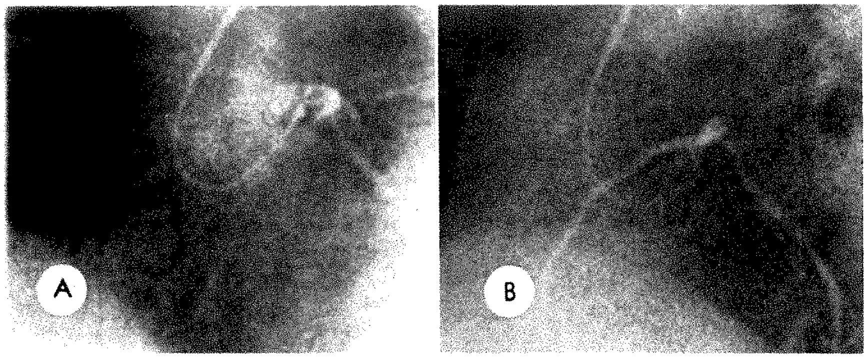

Fig. 2. Selective left coronary arteriography. (*A*) Hand injection. There is incomplete filling of the anterior descending division of the left coronary artery. (*B*) Same patient. With pressure injection, adequate filling of the anterior descending and circumflex divisions of the coronary artery was obtained.

to either side at any time during the procedure. This latter maneuver allows easier intubation with better visualization of the coronary arteries which can be roentgenographed during rotation of the patient.

We plan to compare different methods of roentgenographic recording: cine, rapid film changer and single roentgenograms with magnification (Fig. 4, *A* and *B*).

TECHNIQUE

The right brachial artery is exposed in the antecubital space and is prepared by pre-placing a single, horizontal mattress suture of No. 5-0 silk and puncturing and filling the distal artery segment with 25 mg. of heparin in 5 cc. of isotonic saline solution. Puncture arteriotomy is carried out in the center of the suture space and the catheter is introduced. The percutaneous transfemoral approach may also be used.

The catheter with deflector described above is advanced to the ascending aorta while the electrocardiographic and aortic pressure tracings are constantly monitored. A heparinized glucose solution is slowly infused through the catheter attached by a connector to a pressure injector.*

The Cordis injector allows us to control the amount, duration and electrocardiographic programming (intercalated angiography) of the injection.[3]

Contrast medium under pressure is injected in small volumes in order to visualize the coronary artery orifices. Cannula-

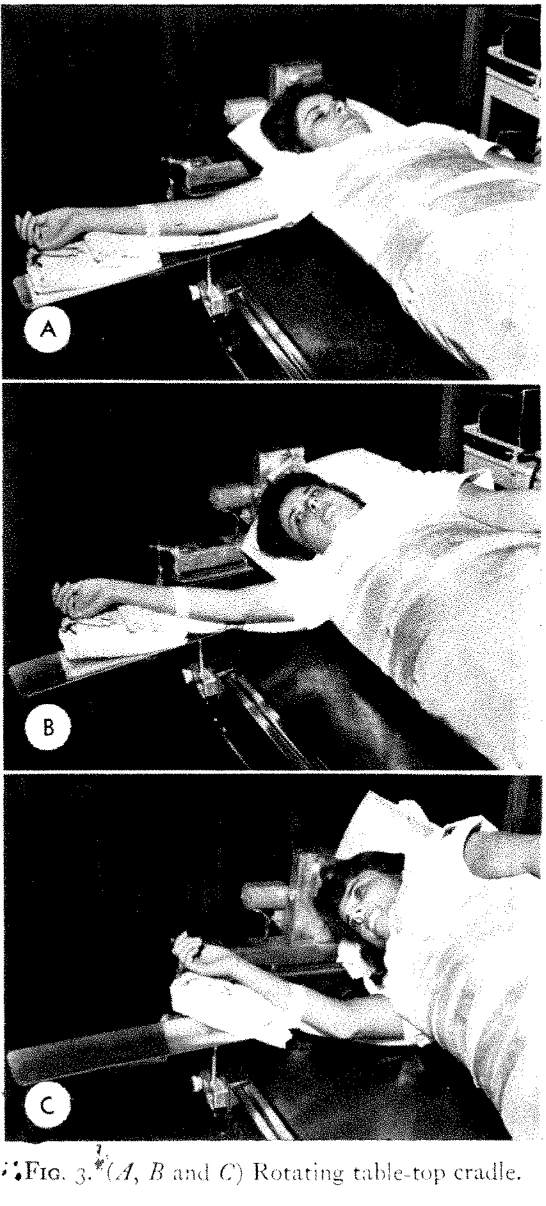

Fig. 3. (*A*, *B* and *C*) Rotating table-top cradle.

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* Manufactured by Cordis Corporation.

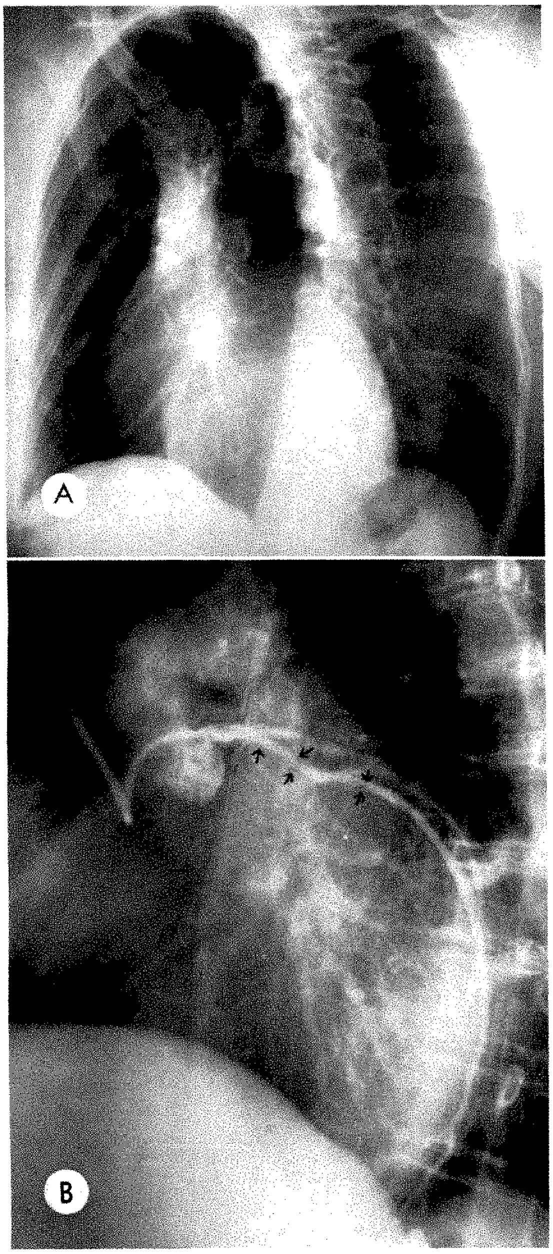

Fig. 4. Selective, programmed, magnified left coronary angiography. (*A*) Conventional left anterior oblique view. (*B*) Same patient. Coronary arteriogram obtained by the technique of roentgenographic magnification. There is obstruction at the origin of the anterior descending division of the left coronary artery and two areas of atherosclerotic narrowing of the circumflex branch (arrows).

tion is accomplished by rotation and manipulation of the deflector tip of the catheter. The procedure is monitored on a television screen. Roentgenography is carried out with the patient at different degrees of obliquity, while being rotated and after administration of a short acting coronary dilator. From 1 to 3 successive injections are performed during each filming sequence.

When selective intubation of the coronary arteries cannot be accomplished, a loop catheter (modified William's[19] catheter) is positioned at the root of the aorta and stuttered injections of 60 cc. of 75 per cent hypaque are made (3 successive injections of 20 cc. of 75 per cent hypaque each, or 2 injections of 30 cc. each, the latter when the ascending aorta is dilated). Each bolus is injected during diastole. The cineangiographic study is performed first and the patient is then quickly positioned by sliding the movable table top over the roll film changer or a cassette for serial or magnified roentgenograms.[17]

The procedure also comprises the recording of left ventricular and aortic pressure curves, right atrium transit time with indocyanine green sinus flush injections to rule out coronary ostial stenosis, and left ventricular and aortic injections, if needed.

DISCUSSION

Adequate interpretation of coronary angiograms depends on the optimum filling of the coronary vessels and on satisfactory registration of the roentgenographic image. The former condition is best fulfilled with selective intubation of the coronary arteries which in our hands has been achieved with a high degree of success with the deflector-tip catheter previously described, combined with pressure injection. The latter condition is accomplished by cinefluorography, mainly for the dynamics, complemented by film changer and single roentgenograms with magnification for high detail and resolution of small arteries. Biplane recording despite the theoretic advantage of obtaining two views with a single injection[18] has been abandoned. It has proved to be too cumbersome and improved radiopaque contrast media allow multiple injections with relative impunity. Moreover, the rotating cradle permits recording of the vessels at several degrees of obliquity during roentgenography.

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ADDENDUM

Our rotating table-top cradle is now being utilized in other procedures such as bronchography and myelography. Another rotating cradle which will rotate 360°, have a lateral shift and will permit examination in the vertical and horizontal positions, is being investigated. Our specially designed catheter has other uses such as the selective intubation of arteries that originate from the aorta and for special venous catheterizations such as after porto-caval shunt operations. A report of the use of this catheter will be made at a later date when more experience is accumulated.

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THROMBOTIC OCCLUSION OF THE ABDOMINAL AORTA*

SEVEN YEAR ANGIOGRAPHIC STUDY OF A CASE

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THE clinical and pathologic aspects of insidious occlusion of the abdominal aorta and iliac vessels have received the continued attention of many investigators during the past 2 decades. Leriche[14,15] in 1923 and 1940 was the first to draw attention to the fact that thrombosis of the aortic bifurcation can occur slowly and progressively and produce only mild symptoms referable to the lower limbs and to sexual function in the male. Indeed, the syndrome now bears his name. Prior to this, it had been assumed that thrombosis was associated with a dramatic onset and was almost invariably fatal.[4] Subsequent studies have amplified Leriche's original observations and have more accurately defined the nature and course of the disease.

With the use of aortography, it has been possible to localize the sites of arterial obstruction. Translumbar aortography was first performed in 1929 by dos Santos *et al.*,[8] who demonstrated that the aorta and its branches could be readily visualized during life. Although Price and Wagner[20] are often cited as the first to use aortography to demonstrate thrombosis of the abdominal aorta, Leriche and Morel[15] utilized this technique in 1 of their cases in 1939. In the past decade translumbar aortography and, more recently, intravenous aortography[25–28] have been used with increasing frequency to delineate the extent of lesions prior to surgery.[1,5,6] Excellent studies have been published with illustrations of the occluded arteries and the numerous compensatory collateral channels.[4,7,19,23] By means of angiography, many investigators have shown the various segments of the abdominal arterial tree that may become affected, thereby producing the Leriche syndrome. The present case provides clinical and roentgenographic documentation of the 7 year course of the disease with confirmation of the roentgenographic findings at necropsy.

REPORT OF A CASE

A 45 year old Caucasian housewife was admitted to The New York Hospital for the seventh time on March 2, 1962, because of a persistent ulcer on the right great toe of 1 month's duration. The present illness began in 1953, with severe low back pain radiating to the buttocks and thighs on climbing a flight of stairs or walking one block. Physical examination at that time revealed absent dorsalis pedis and posterior tibial arterial pulses bilaterally and hyperesthesia of the dorsum of the right great toe. Roentgenographic studies of the abdomen showed calcific deposits in the aorta at the level of the third and fourth lumbar vertebrae. In 1955, the patient was re-admitted because of persistently cold feet and ulceration of the right middle toe. Hip and thigh claudication and paresthesias in the legs associated with walking persisted. At that time, the blood pressure was 120/75 mm. Hg. An abdominal roentgenogram showed a heavy plaque-like density in the wall of the aorta extending from the first to the third lumbar vertebrae with numerous linear calcific deposits in the area of the bifurcation. Translumbar aortograms on December 7, 1955, demonstrated irregular filling defects throughout the aorta below the renal arteries. The origin of the right common iliac artery was also markedly narrowed (Fig. 1). Exploratory laparotomy on December 28,

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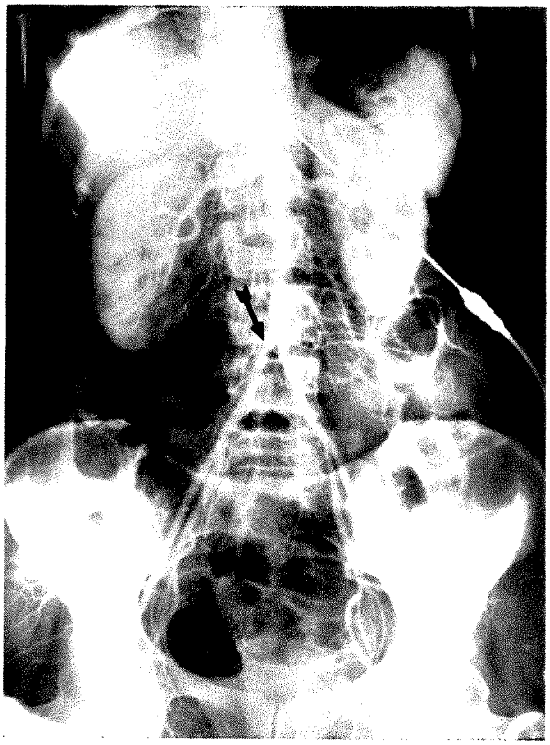

FIG. 1. Translumbar abdominal aortogram made on December 7, 1955, showing intact renal arteries. The lumen of the entire abdominal aorta is narrowed and irregular; the aortoiliac arteries are hypoplastic. Note the marked narrowing of the lumen of the right common iliac artery at its origin (arrow).

1955, confirmed the fact that the lumen of the aorta was narrowed immediately below the renal arteries. Endarterectomy and thrombectomy were peformed, and a cylindrical thrombus and adherent atheromatous material measuring 1.5 by 6 cm. were removed. Postoperatively, both dorsalis pedis and posterior tibial pulses were palpated. The patient remained asymptomatic for only 2 months. Intermittent claudication recurred and became progressively worse during the 4 succeeding years. Of interest was the patient's obstetrical history during this period. Following a full term spontaneous delivery in 1940, and a premature delivery in 1948, she had spontaneous abortions in 1954 and 1956, during the sixth and eighth weeks of gestation, respectively.

At the time of sixth admission, in March 1960, the patient was unable to walk more than 10 steps without severe pain in the legs and buttocks. A painful ulcer on the left great toe was also present. On physical examination the blood pressure was 130/70 mm. Hg. There were diminished pulsations of the right femoral, popliteal, dorsalis pedis, and posterior tibial arteries; only a weak femoral pulse was palpated on the left; no trophic skin changes were noted. A harsh, systolic bruit (Grade 2-3) was heard over the abdomen extending from the umbilicus to the groin. Intravenous aortograms on March 10, 1960, showed further narrowing and irregularity of the aorta below the renal arteries with abrupt constriction of the lumen immediately above the bifurcation (Fig. 2). Numerous filling defects were evident, both at the bifurcation of the aorta and in the proximal segments of the common iliac arteries. Surgery was advised on the condition that the patient would first cease smoking. She continued to smoke 10–20 cigarettes a day and was discharged without having an operation.

During the next 2 years, the patient's arterial insufficiency became even more incapacitating, and ulceration of the right great toe finally necessitated her seventh admission to the hospital in March, 1962. In addition, mild dyspnea on exertion and angina pectoris had begun 18

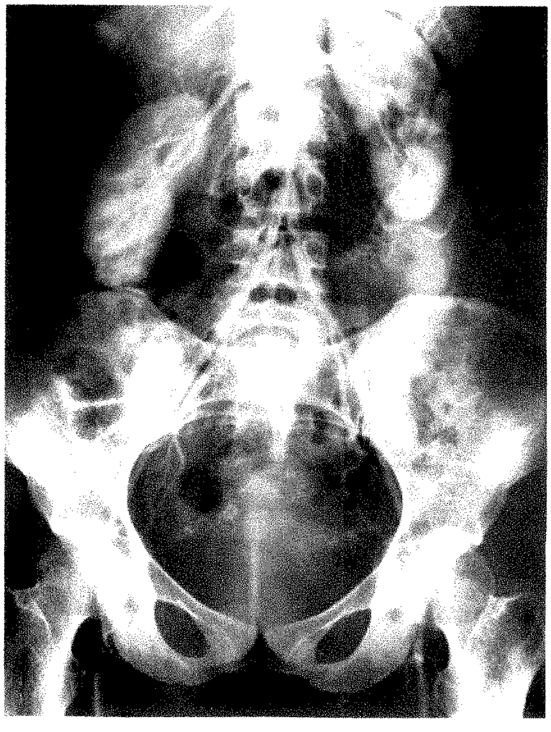

FIG. 2. Intravenous abdominal aortogram made on March 10, 1960, five years after endarterectomy. Again, there is marked narrowing and irregularity of the lumen of the abdominal aorta.

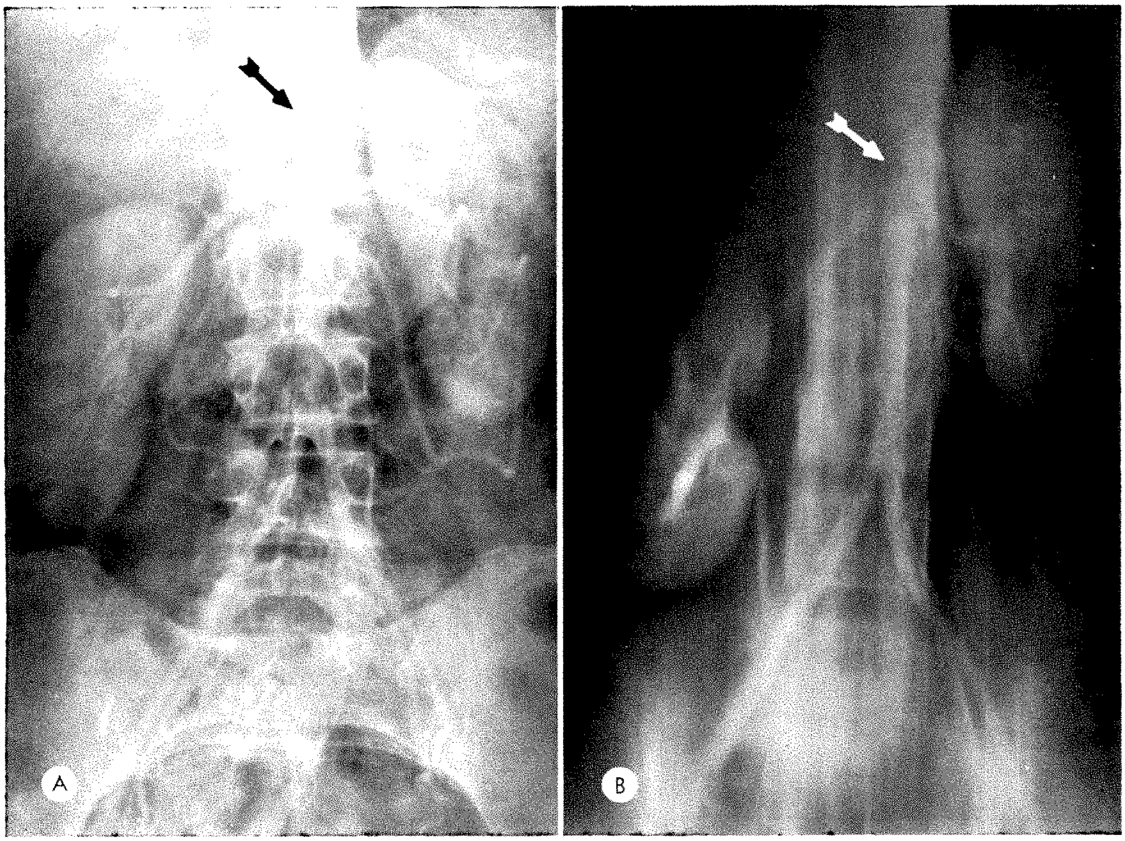

Fig. 3. Intravenous abdominal aortograms made on March 5, 1962. (*A*) Abdominal aortogram now shows a filling defect (thrombus) above the right renal artery (arrow). (*B*) Aortotomogram more clearly outlines the thrombus above the right renal artery (arrow). Note linear calcification along the lower borders of the aortoiliac system, above and below the abdominal aortic bifurcation.

months earlier. On physical examination, the blood pressure was 150/80 mm. Hg, and the pulse was 84 per minute and irregular. A nonradiating (Grade 2) systolic murmur was heard over the third intercostal space at the left sternal border. There were markedly diminished femoral pulses, and vascular pulsations were absent in the lower extremities. The skin of both legs was dry and scaly and there was a 3 mm. in diameter, dark, necrotic eschar on the plantar surface of the right great toe. The serum cholesterol was 252 mg. per 100 ml., the fasting sugar was 68 mg. per 100 ml., and the blood urea nitrogen was 12 mg. per 100 ml. An electrocardiogram showed no abnormalities. Intravenous abdominal aortograms, aortotomograms and peripheral arteriograms on March 5, 1962, showed abrupt narrowing of the aortic lumen immediately above the right renal artery. The remainder of the abdominal aorta was markedly irregular and narrowed with another constriction of the lumen about 2 cm. above the bifurcation. Endarteritic changes were also marked in the iliac arteries (Fig. 3, *A* and *B*). Calcific plaques were noted in the right femoral artery at the level of the mid-thigh.

Exploratory laparotomy on March 9, 1962, showed the aorta to be almost completely occluded and without evident pulsation below the renal arteries. A laminated thrombus with a portion extending beyond the origin of the renal arteries was excised in its entirety. A teflon prosthesis was then anastomosed proximally to the aorta and distally to the left external iliac and right common iliac arteries. The insertion of the prosthesis necessitated ligation of an accessory renal artery that supplied the inferior pole of the left kidney. Bilateral lumbar sympathectomies were also performed.

On the first postoperative day, a strong femoral pulsation was present in the left groin but the right femoral pulse was weak and the skin remained cool; anticoagulant therapy was begun. Despite improvement in the patient's condition, the hematocrit decreased from 41 to 31 per cent on the third postoperative day. The prothrombin time was 60 seconds. Accordingly, anticoagulants were discontinued and vitamin K was administered; the patient was also transfused. On the following morning the hematocrit was 40 per cent. At that time the operative wound was healing well and no abdominal masses were palpable. The femoral pulses were present bilaterally and both feet were warm. The next day the patient developed a nonproductive cough, and wheezes and ronchi were heard over the anterior and lateral left hemithorax. A roentgenogram of the chest showed patchy infiltrates in the left lung and at the right base. Despite antibiotic therapy, her condition became worse and rales were heard in the chest bilaterally; the temperature rose to 38.4° C. and the white blood cell count was 17,400/mm.^{3} with a shift to the left. Subsequently, she developed paralytic ileus, gastric dilatation, vomited bile-stained material, and died suddenly on the ninth postoperative day.

Findings at necropsy. The body was that of a well developed and nourished female measuring 156 cm. in length and weighing 46 kg. There were circular, dry ulcers measuring 1 cm. in diameter on the right great toe and on the posterior aspect of the left heel. The abdomen contained a partially healed, 32 cm., midline incision. The heart weighed 325 gm.; the left and right ventricles, at the base, measured 14 and 3 mm. in thickness, respectively. The lumina of the coronary arteries were focally narrowed up to 50 per cent by atheromatous plaques. The lungs together weighed 1,189 gm. The pleural surfaces were smooth, and the cut surfaces were moderately hyperemic, exuding a pink frothy fluid. Microscopically, the alveolar septa were edematous and the capillaries were engorged; the alveoli contained edema fluid and macrophages, some of which were vacuolated and filled with hemosiderin pigment.

A large, partly organized, retroperitoneal hematoma (500–800 ml.) surrounded a portion of the abdominal aorta, the aortic prosthesis, inferior vena cava, iliac vessels, ureters, the midportion of the sigmoid colon and the adrenal glands. The iliac and renal veins and the inferior vena cava were patent and there was no evidence of ureteral or intestinal obstruction by the hematoma.

The aorta had been transected 3 cm. distal to the renal arteries, and a Y-shaped teflon prosthesis had been anastomosed to the descending aorta and the right common iliac and left external iliac arteries. All anastomoses were patent and intact; a specific bleeding point was not identified. Immediately proximal to the renal arteries, the aorta was slightly dilated. The lumen of the aorta then became narrowed to 60–70 per cent of its original diameter by a series of raised, rough, grumous, mural plaques and mural thrombi along the right border (Fig. 4*A*). Distal to the renal arteries, the lumen was also markedly narrowed by generalized atheromatous thickening of the wall. In the aortic segment of the bypassed vessel, an old thrombus was superimposed upon confluent, fibrofatty, and calcific plaques and grumous material; the diameter of the lumen did not exceed 1.2 cm. at any point. The origin of the left common iliac artery was partly occluded by similar material, whereas the right common iliac artery was completely obstructed by a recent, dark, red, partially organized thrombus. There was a moderate degree of atheromatous change in the wall of the thoracic aorta which microscopically showed moderate fibrofatty intimal thickening. The changes were pronounced in the abdominal aorta and common iliac arteries, where the media was thin and replaced by fibrous tissue admixed with a few chronic inflammatory cells; the intima was markedly thickened and contained amorphous, fatty and calcific deposits as well as an increased amount of collagen (Fig. 4, *B* and *C*).

The kidneys together weighed 280 gm. The left kidney contained a 1 by 6 cm., discrete, yellow, soft infarct with a hyperemic border which microscopically showed coagulative change, hyperemia, and a few neutrophils. The kidneys were otherwise normal in the gross, but microscopically there was moderate intimal proliferation in large arteries and marked narrowing of the lumina of arterioles by intramural hyaline deposits. Cultures of the blood did not yield organisms; *Proteus*, *Staphylococcus aureus* and *Streptococcus* gamma were isolated from the lower lobe of the right lung.

DISCUSSION

In 1940, Leriche[15] described a syndrome, occurring in middle-aged males, which is the result of insidious thrombotic occlusion of the aorta at the bifurcation. It is characterized by extreme fatiguability, symmetric

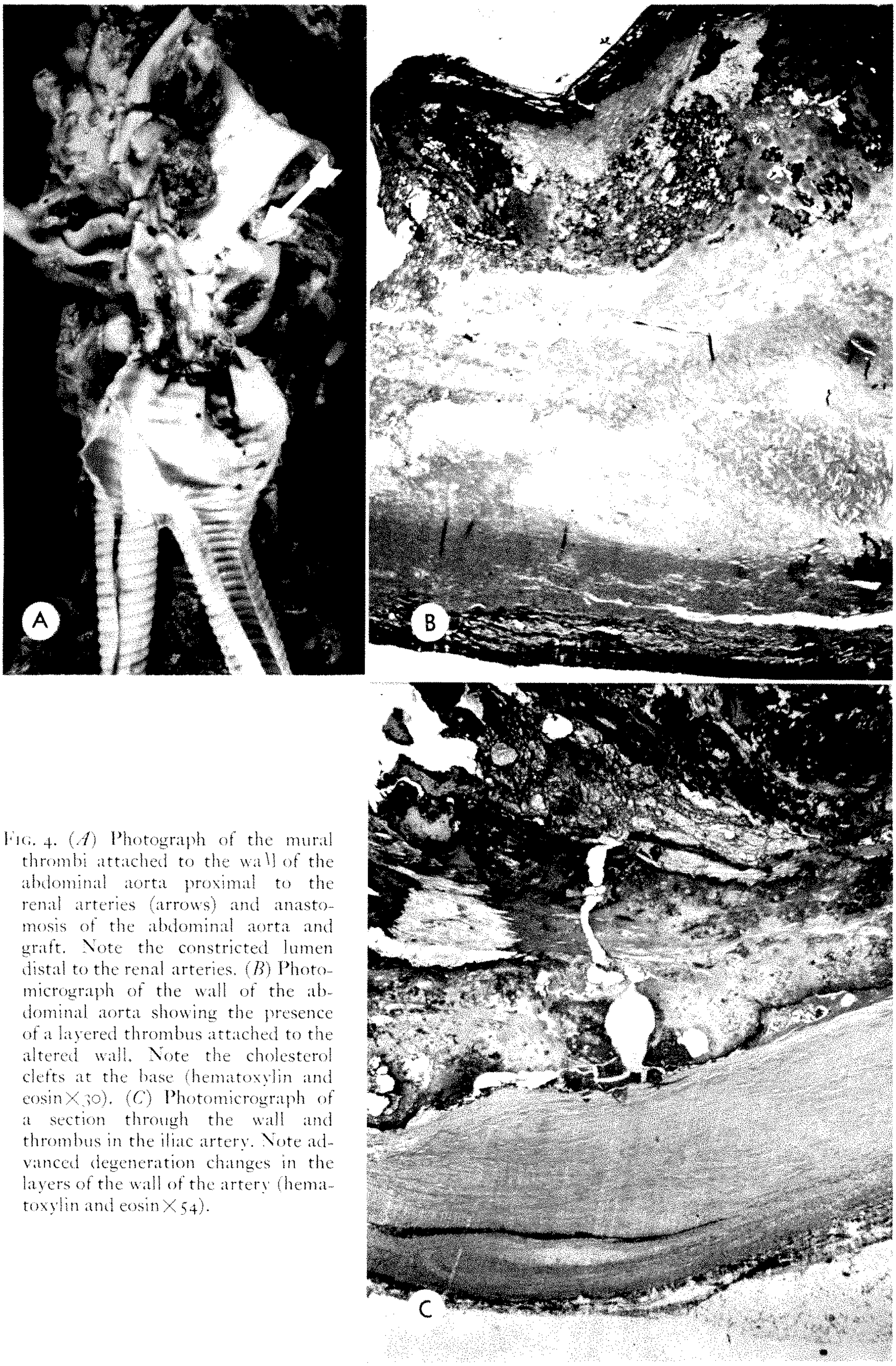

FIG. 4. (*A*) Photograph of the mural thrombi attached to the wall of the abdominal aorta proximal to the renal arteries (arrows) and anastomosis of the abdominal aorta and graft. Note the constricted lumen distal to the renal arteries. (*B*) Photomicrograph of the wall of the abdominal aorta showing the presence of a layered thrombus attached to the altered wall. Note the cholesterol clefts at the base (hematoxylin and eosin × 30). (*C*) Photomicrograph of a section through the wall and thrombus in the iliac artery. Note advanced degeneration changes in the layers of the wall of the artery (hematoxylin and eosin × 54).

muscular atrophy of the lower extremities, absence of nutritional changes in the skin and nails, pallor of the legs and feet even when standing, sluggish healing of the wounds in the lower limbs, and the inability to maintain a stable penile erection. He also noted that the arterial pulsations are absent in the legs and that the systemic blood pressure, as measured in arms, is slightly elevated. The over-all incidence of thrombotic occlusion of the aortic bifurcation at autopsy is 0.12–0.15 per cent,[24,29] while the incidence in a series followed at a vascular clinic was 0.61 per cent.[18] In each of 3 large series, where 65, 47, and 105 patients were studied, there was a male preponderance of about 8:1 and the peak incidence was in the sixth decade.[2,7,17]

The presence of vascular hypertension in patients suffering from aortoiliac obstruction is notable. While Leriche suggested that slight elevation in the blood pressure was to be expected in the course of the disease, most observers have found that hypertension occurs in less than half the patients with this disorder.[2,18,24] The etiology of the hypertension associated with thrombotic occlusion of the abdominal aorta has been a subject of considerable discussion. Leriche[15] believed that elevation of the blood pressure occurred in the absence of renal involvement, presumably secondary to arterial obstruction alone. In support of this explanation, Horwitz[12] reported a case of isolated, acute aortoiliac obstruction wherein the blood pressure rose from 105/75 to 170/124 mm. Hg. On the other hand, Brotchner[3] found that acute occlusion of the aorta below the renal arteries in dogs caused a rise in pressure of only 8.8 mm. Hg, suggesting that aortoiliac obstruction has little effect on the blood pressure. That many patients with complete occlusion of the abdominal aorta show no significant elevation of blood pressure strongly supports the latter conclusion. Straus *et al.*[29] attributed the associated hypertension to renal ischemia resulting from occlusion of the renal arteries, present in all 3 of their patients. Wyatt and Felson[31] have shown roentgenographically in 3 patients that retrograde propagation of a thrombus with partial obstruction of the flow of blood into one renal artery corresponded with the sudden onset of marked hypertension which was relieved in all the cases by nephrectomy. However, Starer and Sutton[24] reported 5 cases where there was associated renal artery stenosis; 2 of the 5 had normal blood pressures. Over the course of 7 years, our patient's blood pressure rose from 120/75 to 150/80 mm. Hg; the pressure change was largely systolic in nature and of no clinical significance. In spite of moderate to marked narrowing of the renal arteries, the rise in the systolic blood pressure may be attributed to atherosclerosis of the aorta with partial occlusion of the lumen.[15] Significant hypertension, when it occurs in association with this disorder, is probably a manifestation of systemic vascular disease and does not appear to be directly related to the degree of involvement of the renal arteries except in isolated cases.

Following Leriche's original description of the syndrome, a number of investigators have studied the disease, pointing out that many additional signs and symptoms are frequently associated with aortoiliac obstruction. Some of these were present in our patient. Intermittent claudication of the hip has been emphasized by De Wolfe *et al.*,[7] who found that 89 per cent of 47 patients had this symptom. The pain frequently radiated to the low back, thigh, and leg. Similarly, Beckwith *et al.*[2] noted hip claudication in 63 per cent of their patients. In fact, De Wolfe *et al.*[7] stated that intermittent claudication of the hip or buttocks is pathognomonic of an arterial occlusion above the inguinal ligament. Beckwith *et al.*[2] reported that 86 per cent of their patients exhibited diminished skin temperature in the legs, and atrophy of the skin and subcutaneous tissues was present in 48 per cent of their cases. The trophic changes are probably due to obstruction within the external iliac arteries; the patients described by De Wolfe *et al.*,[7] who had occlusion of the aorta or common iliac arteries alone had no evidence of peripheral trophic lesions. Abdominal murmurs (bruits) are heard with variable frequency. Massarelli and Estes[17] stated that systolic abdominal murmurs were present in only 12 of 105 patients. However, they were of the opinion that bruits were overlooked in a great number of their cases and that this physical finding was probably present in every patient suffering from aortoiliac occlusion. Starer and Sutton[24] studied 10 patients with particular reference to murmurs and observed that a systolic bruit was evident in only 4. The murmur was attributed to blood-current eddies produced by the irregularly stenotic arterial channels. According to Edwards,[9] when a murmur is present, angiography always demonstrates a partially patent lumen even though there may be no apparent pulse. In general, the murmur is loudest when the obstruction is severe but not complete.

Although the etiologies of spontaneous abortion are many, it is of interest that the patient described above aborted spontaneously on 2 instances during the course of her disease. One can only speculate that arterial insufficiency involving the genital tract may have been responsible for termination of the pregnancies. This, then, would represent the counterpart reaction in the female and match the inability to maintain a stable erection in the male.

The precise nature of the disease process which results in aortoiliac occlusion has been the subject of discussion. Leriche[14,15] originally suggested that the disorder was primarily inflammatory in nature. This thesis is well summarized by Shapiro[23] as follows: (1) thrombotic obliteration of the aorta is relatively rare and often begins in the iliac arteries, whereas atherosclerosis is more common in the terminal aorta; (2) the disease occasionally occurs in young people without evidence of other atheromatous arterial disease; (3) cases have been reported where diffuse arteritis was present; and (4) severe periarteritis involving veins, lymphatics, and sympathetic ganglia has been recorded. Other etiologic factors such as trauma, dissecting aneurysm, and osteophytic irritation of the aorta have been reviewed by Starer and Sutton.[24] Rarely, thrombotic occlusion may occur in the absence of underlying disease.[10] Although many conditions may give rise to the aortoiliac obstruction, the disorder is generally secondary to atherosclerotic involvement of the terminal aorta and common iliac arteries.[2,4] In the patient described above atherosclerosis was a prominent feature of her disease; there was no evidence of arteritis.

The insidious nature of the disease affecting the terminal segment of the aorta and the iliac arteries is illustrated by the case herein presented. Over the course of 9 years, the patient suffered from progressive arterial insufficiency extending from the buttocks and hips distally to the feet. That endarterectomy and thrombectomy, after 2 years of clinical disease, were successful in relieving her symptoms for a 2 month period strongly suggests that the predominant lesion was located in the aorta and iliac arteries at that time. Roentgenographically, the progress of the disease was followed over an 8 year period. The calcific densities immediately above the bifurcation of the aorta (calcified atheromata) were the initial roentgenographic manifestation of the disease. These were followed, within a year, by a more proximal heavy plaque-like density along the right border of the aorta and numerous linear calcific densities at the bifurcation, suggesting progression of the process in both directions. Translumbar aortography in 1955 (Fig. 1), demonstrated involvement of the distal aorta and common iliac arteries. Further narrowing of the involved vessels was evident in 1960 (Fig. 2) and by 1962 (Fig. 3, *A* and *B;* and 4*A*), the disease process had extended above the renal arteries with dilatation of the proximal aorta. While there was no evidence of complete occlusion of the vessels roentgenographically, the symptoms, abdominal aortograms, and findings at autopsy are all indicative of severe, progressive compromise of the arterial lumina by continual disease process.

Numerous studies of patients with the Leriche syndrome have shown that the site of complete arterial occlusion is variable. Leriche and Morel[16] believed that thrombotic occlusion usually began in 1 of the common iliac arteries, extending proximally into the aorta and ultimately obstructing the flow into the contralateral common iliac artery at the aortic bifurcation. On the other hand, less frequently, thrombosis was observed to begin in the aorta itself. In the 26 cases studied roentgenographically by De Wolfe *et al.*,[7] the arterial block existed within the abdominal aorta in 7, within one common iliac in 11, and within both common iliacs in 4, while, in the remaining 3 cases, 2 or more occluded segments were even more widely separated. Thus, the initial obstruction may occur at any site, with secondary proximal and distal progression. Starer and Sutton[24] believe that the distal limit of obstruction is determined by the collateral circulation that is able to carry blood into the iliac arteries and produce a sufficiently rapid flow to prevent thrombosis. The atherosclerosis and thrombosis in this case extended above the renal arteries (Fig. 3, *A* and *B;* and 4*A*). Numerous instances of partial or complete renal arterial obstruction have been recorded.[24,29,31]

Complete thrombotic occlusion may occur through a number of mechanisms. Burt *et al.*[4] state that thrombosis usually begins in the common iliac arteries, because of the small diameter of the vessels, and extends proximally. They attribute thrombosis to diminished flow, resulting from compromise of the diameter of the lumen. In addition, these authors suggest that in some instances a thrombus formed on an atheromatous plaque in a more proximal segment of the aorta may be the source of an embolus which lodges at the bifurcation or within 1 of the iliac arteries. The factors responsible for thrombotic occlusion are: constriction of the vessels by atheromatous plaques, erosion of plaques, and diminution of blood flow. These may be operant in any segment of the arterial tree. The proximal propagation of thrombi, occurring on 2 occasions in this case, may be readily understood when one considers the extensive lesions in the vessel wall and the marked reduction in blood flow due to distal narrowing of the lumen. By this mechanism, hemostatis, thrombosis, and occlusion occur. On the other hand, the same conditions (stasis and disease of the vessel wall) prevail in the iliac arteries where the constriction is proximal, thereby explaining the high frequency of occlusion below the aortic bifurcation.

In the initial studies by Leriche,[15] thrombotic occlusion of the aortic bifurcation was considered to be an isolated disease process that affected only the terminal aorta and the common iliac arteries. However, clinically this patient complained of mild dyspnea on exertion and angina pectoris late in the course of the disease. At autopsy, there was moderate to marked narrowing of the lumina of the coronary and renal arteries, and of the thoracic aorta, demonstrating the systemic distribution of the disease. Beckwith *et al.*,[2] who did thorough studies on 65 patients, found evidence of heart disease in 75 per cent, renal disease in 51 per cent, and cerebrovascular disease in 21 per cent. Twenty-two of their patients died; 2 because of thrombotic occlusion of the renal arteries, and the remainder "usually" because of disease of the coronary, renal or cerebral vessels. Massarelli and Estes[17] noted that 83 per cent of the deaths in their series were related to atherosclerosis of the coronary or cerebral arteries. Most investigators believe that there is an underlying generalized atherosclerotic process in cases of aortoiliac thrombosis. It has been pointed out that atherosclerosis of the abdominal aorta usually begins and advances at a much younger age than is commonly appreciated. Zinserling[32] observed aortic lesions, characteristic of early atherosclerosis, in 95 per cent of 320 subjects under the age of 16 years. Similarly, Holman *et al.*[11] noted that patients 3 years of age and older had at least minimal sudanophilic intimal deposits in the aorta. The aortic ring was the first region of the vessel to show fatty streaks; however, it was in the abdominal portion of the aorta that the distinctive pattern of increasing lesions was manifested with advancing age of the subject (8 to 18 years). Fibrous plaques were most numerous in the abdominal portion. In a study of males who had reached the age of 35 years, the 45 per cent of the intimal surface of the abdominal aorta was covered with fatty streaks and fibrous plaques; in females of the same age, the area involved was only 5 to 7 per cent less. Roberts *et al.*[21] have shown that from the ages of 20 to 60 years there is a more rapid progression of atherosclerosis in the abdominal aorta and common iliac arteries than any other vessels. The above findings are significant because they document the degree of atherosclerosis of the abdominal aorta that is present in presumably normal, middle aged adults. It is noteworthy that the peak incidence of the Leriche syndrome occurs between the ages of 50–59 years, which correlates well with the rapid phase of arterogenesis in the aorta and iliac arteries. In their study of 36 patients suffering "aortic catastrophies" (aneurysm or thrombosis), Roberts *et al.*[22] observed that the atherosclerotic change in the abdominal aorta was moderately more severe than in age-matched controls. Also of note is that the former group had more severe generalized atherosclerosis. These findings strongly suggest that the aorta and iliac arteries of younger patients who do have rapidly progressive disease are more susceptible to atherosclerosis.

Although the etiology of atherosclerosis is unknown, serum cholesterol levels, hypertension, and smoking are currently considered to be significant factors in the initiation and progression of the disease. Theis[30] reports that 62 per cent of his patients with aortoiliac thrombosis had serum cholesterol levels above 300 mg. per cent. On the other hand, Massarelli and Estes[17] noted that only 29 per cent of the 80 cases they studied had serum cholesterol levels greater than 250 mg. per cent. Hypertension is probably of little importance in the etiology of this disorder for, as mentioned previously, less than half of the cases have elevated blood pressures. To our knowledge, smoking has not been studied specifically in patients with the Leriche syndrome. Juergens *et al.*[13] report that the incidence of smoking in patients with arteriosclerosis obliterans (femoral artery occlusion—296 cases; aortoiliac occlusion—224 cases) was 97.5 per cent, compared with a control figure of 74 per cent. However, the incidence of arteriosclerosis in patients smoking more than 20 cigarettes a day was no different from that in the control group. Further study of these variables is necessary before etiologic significance can be ascribed to them.

SUMMARY AND CONCLUSIONS

A 45 year old woman suffering from insidious thrombotic occlusion of the aorta was followed over a 9 year period with angiographic demonstration of progression of the disease. Necropsy confirmed the clinical and roentgenographic findings of severe atherosclerosis of the terminal aorta and iliac arteries; moderately advanced sclerosis of the renal and coronary arteries was also present.

The syndrome of abdominal aortic occlusion, originally described by Leriche, has been amplified by subsequent investigators who have noted the frequent association of claudication localized around the hip and buttocks, abdominal murmurs, and trophic changes in the skin and subcutaneous tissues of the legs. Hypertension is occasionally present in patients with this syndrome; rarely, thrombotic obstruction of one of the renal arteries can also be demonstrated. Progression of the atherosclerotic process is insidious. Ultimate cardiac, renal, or cerebral manifestations of disease at a relatively young age reflect the systemic nature of vascular lesions in patients with this disorder. Atherosclerotic changes in the abdominal aorta usually begin at a very early age with more rapid progression of the disease between the ages of 20 and 60 years; aortoiliac thrombosis has its peak incidence between 50 and 59 years. These findings strongly suggest that the aorta and iliac arteries of younger patients who do have rapidly progressive disease are especially susceptible to atherosclerosis. Aortoiliac occlusion is uncommon in the absence of advanced systemic arterial disease.

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AORTIC ATRESIA WITHOUT SIGNIFICANT HYPOPLASIA OF THE ASCENDING AORTA

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AORTIC atresia, the most common cause of cardiac death in the first week of life, has received sufficient attention in the literature so as to suspect this cardiac anomaly clinically. The history of cardiac failure during the first week of life, together with the physical findings, roentgenograms and electrocardiograms, allow the ante mortem diagnosis in a high percentage of cases.

In atresia of the aortic valve, the diminished blood flow in the ascending aorta consists only of retrograde flow to the coronary arteries. Consequently, in this condition the ascending aorta is usually of diminutive size and not much larger than the coronary arteries themselves; this is considered characteristic of the condition. It is the purpose of the author to describe 2 cases of aortic atresia in which the ascending aorta was of essentially normal caliber.

REPORT OF CASES

Case i. This full term baby boy was admitted to the University of Minnesota Hospitals on March 12, 1963 at 4 days of age because of congestive heart failure since the second day after birth.

Physical Examination. The patient was mildly cyanotic and in mild respiratory distress. The pulse rate was 128 per minute and regular; the respiratory rate was 65 per minute. Pertinent findings were a Grade iii/vi systolic murmur heard over the entire precordium but mostly in the aortic area, a liver edge felt 4 cm. below the right costal margin, and very weak radial and femoral pulses. The electrocardiogram showed normal axis with left ventricular hypertrophy and strain.

Roentgen Findings. Cardiac fluoroscopy on admission revealed cardiomegaly with increased pulmonary vascularity resembling passive congestion and absence of left atrial enlargement (Fig. 1, *A* and *B*). Forward angiography showed dilution of contrast medium at the right atrial level and early filling of the descending aorta through a reversing patent ductus arteriosus. The findings were considered to be consistent with aortic atresia which was confirmed by aortography via left brachial artery (Fig. 2, *A* and *B*). The main pulmonary artery was faintly filled through a reversing patent ductus arteriosus as indicated by nonfilling of the peripheral pulmonary arteries; there was characteristic dense and prolonged filling of the ascending aorta with no dilution by blood during systole. Because of the demonstration of 3 normally shaped aortic valves and the large size of the ascending aorta, the roentgenologic diagnosis of aortic atresia was doubted by the referring clinician.

Postmortem Findings. Postmortem examination showed: (1) aortic valve atresia; (2) large patent ductus arteriosus; (3) atrial septal defect; (4) right atrial and right ventricular hypertrophy; (5) endocardial fibroelastosis of the left ventricle; (6) hepatomegaly secondary to congestive heart failure; and (7) congestion of both lungs.

Case ii. This 4 day old baby boy, J.K., was admitted on June 30, 1963 with dyspnea and cyanosis. On the day after birth the patient developed progressively increasing tachypnea and cyanosis, especially when crying.

Physical Examination. The blood pressure was 60-50 mm. Hg in arms and legs, the respiratory rate was 120 per minute and weak femoral pulses were palpable bilaterally. A Grade ii/vi systolic murmur was heard at the left sternal border in the second and third left intercostal spaces, which was transmitted to the back. The second sound over the aortic area was decreased. The lung fields were clear to percussion and auscultation. The liver and spleen were palpable two finger breadths below their respective costal margins.

Roentgen Findings. Heart roentgenograms (Fig. 3, *A* and *B*) at the time of admission

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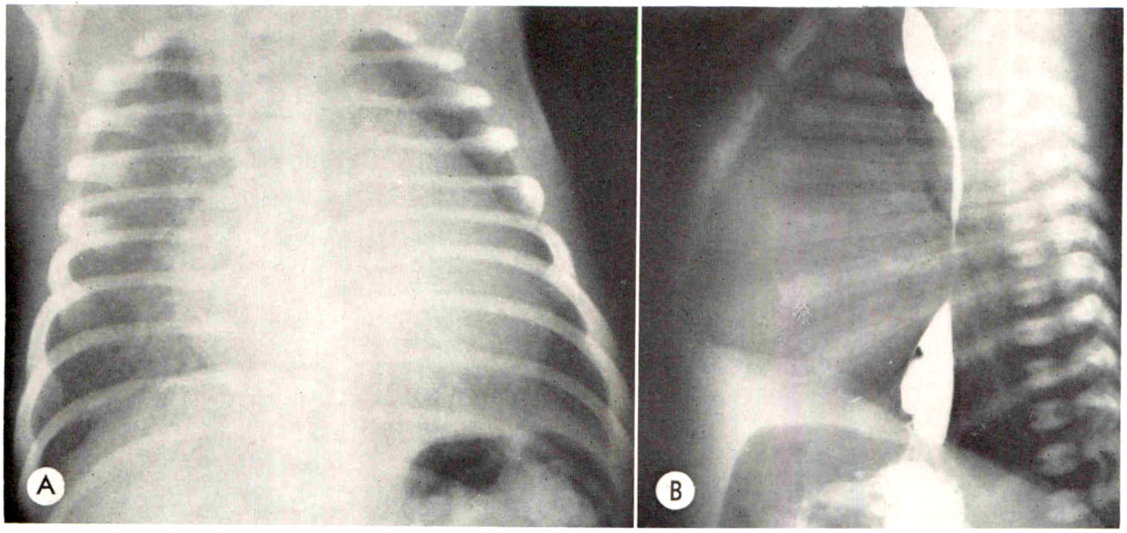

Fig. 1. (*A*) Anteroposterior and (*B*) lateral roentgenograms showing marked cardiomegaly with increased pulmonary vascularity suggesting passive congestion. Note prominent right atrial contour and lack of left atrial enlargement.

showed marked cardiomegaly with increased arterial pulmonary vasculature and a suggestion of left atrial enlargement. Aortography via right brachial artery revealed faint filling of the main pulmonary artery by an apparently reversing patent ductus arteriosus (Fig. 4*A*). An unusually dense filling of the ascending aorta which persisted following clearing of the aortic arch and descending aorta of contrast medium (Fig. 4*B*) was felt to be diagnostic of aortic valvular atresia notwithstanding the fair size of the ascending aorta.

Despite intensive medical treatment, the patient expired 8 days after admission.

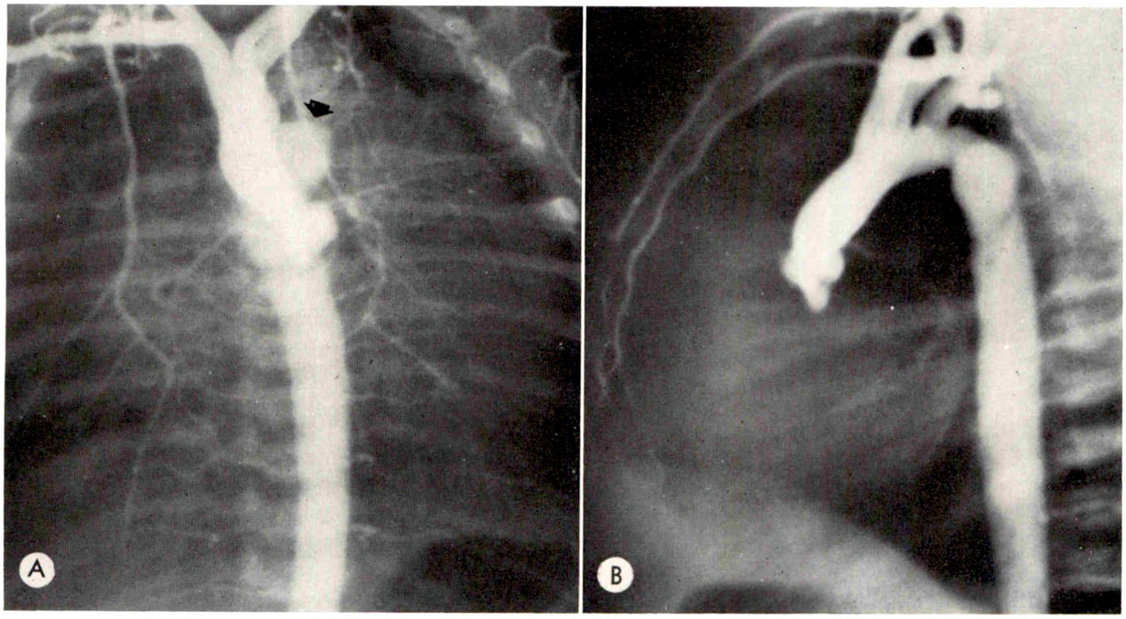

Fig. 2. (*A*) Anteroposterior and (*B*) lateral retrograde aortograms via left brachial artery show dense filling of the entire ascending aorta and filling of the coronary arteries. There are demonstrated three well formed aortic cusps in the lateral view. Minimal filling of the pulmonary artery through the patent ductus arteriosus (arrow) is seen.

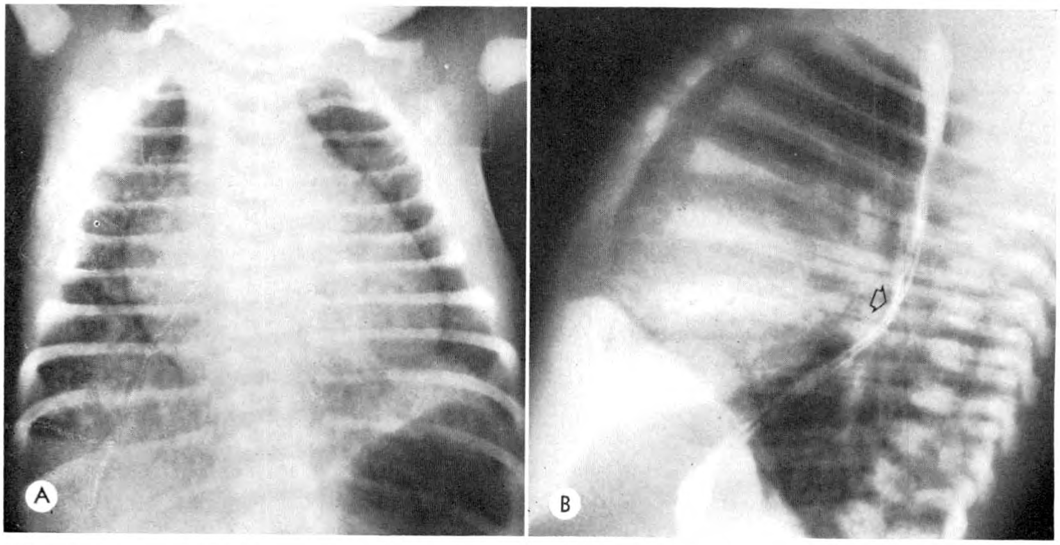

Fig. 3. (*A* and *B*) Marked cardiomegaly and increased pulmonary flow rather than congestion are demonstrated. Note the slight left atrial enlargement (arrow).

Postmortem Findings. Postmortem examination showed: (1) aortic atresia associated with a hypoplastic left ventricle and mitral valve; (2) endocardial fibroelastosis of the left ventricle; (3) atrial septal defect; (4) dilated right atrium, right ventricle and main pulmonary artery; and (5) passive congestion of the liver and kidneys.

DISCUSSION

HEMODYNAMIC AND ANATOMIC FINDINGS

In aortic valvular atresia, the right ventricle not only supplies the pulmonary circulation but also the peripheral circulation by a large reversing patent ductus arteriosus which is invariably present. Since no blood is ejected from the left ventricle into the ascending aorta, there is retrograde blood flow through the ascending aorta to the coronary arteries via the patent ductus arteriosus. The blood flow determines largely the size of blood vessels; therefore, the ascending aorta is characteristically minute since it carries only the coronary flow as compared with the total peripheral circulation in the normal heart. The pulmonary arteries and pulmonary veins, on the other hand, are enlarged due to increased pulmonary flow and venous congestion. An element of venous obstruction is invariably present at the foramen ovale level which has to be forced open in order to allow the necessary left-to-right shunt at the atrial level. Characteristically, in aortic valvular atresia, there is hypoplastic development of the left side of the heart including the left atrium, mitral valve, left ventricle and ascending aorta.

ROENTGENOGRAPHIC FINDINGS

The clinical history of heart failure during the first week of life, mild cyanosis, weak peripheral pulses and right ventricular hypertrophy shown on the electrocardiogram should alert the radiologist to the fact that he may be dealing with a nonfunctioning left ventricle syndrome. The rudimentary left-sided development may be due to severe aortic stenosis, aortic atresia or mitral atresia. The plain roentgenograms usually show considerable cardiomegaly with prominence of the right atrial segment and engorged pulmonary vessels. The pulmonary vascularity may resemble venous engorgement as in Case I or increased pulmonary flow as in Case II. If the obstructive element at the foramen ovale level is very pronounced, pulmonary blood flow may not be considerably increased and the heart may be smaller, resembling somewhat

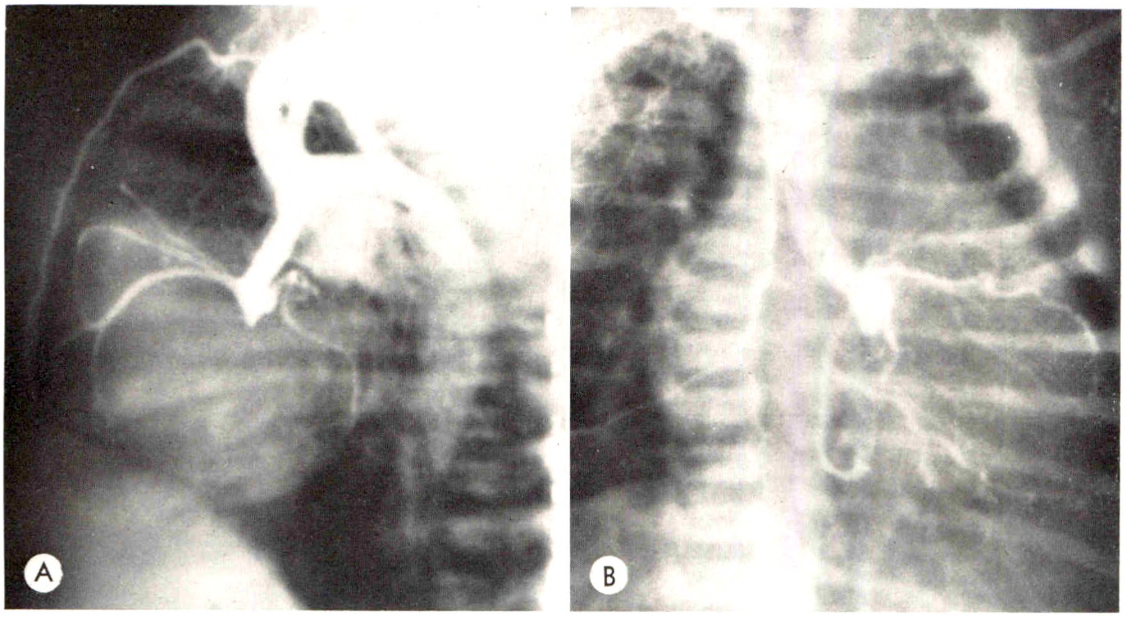

FIG. 4. (*A* and *B*) Unusually dense filling of the entire ascending aorta including the coronary arteries is achieved by "flush" aortography via the right brachial artery. Note persistent opacification of ascending aorta and coronary artery. *B* shows the pathognomonic lack of blood flow through an atretic aortic valve.

conditions associated with venous obstruction such as cor triatriatum or anomalous pulmonary return with venous obstruction. The element of venous congestion is helpful in differentiating this condition from transposition of the great vessels, truncus arteriosus, total anomalous venous return and other conditions of cyanosis associated with increased pulmonary flow. The routine roentgenograms of both our patients were, therefore, consistent with the hypoplastic left ventricle syndrome except for the suggested left atrial enlargement (usually absent in this syndrome) found in Case II.

ANGIOGRAPHIC FINDINGS

The forward angiograms are usually of poor quality since the contrast medium is markedly diluted in the enlarged right atrium which receives the total pulmonary and systemic venous return similarly to total anomalous drainage. Contrary to this latter condition which may well be considered on routine roentgenograms, there is never a right-to-left shunt at the atrial level as is invariably present in total drainage. The enlarged pulmonary arteries usually fill poorly due to dilution of contrast medium and there is filling of the descending aorta through a reversing patent ductus arteriosus. Sometimes, a diminutive ascending aorta may be seen, but commonly the ascending aorta cannot be identified on the forward angiograms. Dilution at the right atrial level without a right-to-left shunt, an enlarged normally placed pulmonary artery and filling of the descending aorta through a reversing patent ductus arteriosus are the salient features.

Of much more diagnostic value is counter brachial aortography via the left or, preferably, the right artery. Generally speaking, the retrograde forceful injection of contrast medium into the brachial artery results in more or less complete opacification of the ascending aorta, depending not only on technical factors, but mostly on aortic blood flow and, consequently, cardiac output. Patients with poor cardiac output and cardiac failure show characteristically dense aortograms with good opacification of the ascending aorta. Nevertheless, during systole blood ejected through the aortic valve tends to immediately "wash out" the contrast medium from the ascending aorta. In aortic valvular atresia, usually excellent opacification of the ascending aorta and even visualization of the aortic valve and coronary arteries, which are almost never seen by this technique, are present. Furthermore, and most significantly, the washout of contrast medium occurs first in the distal arch and descending aorta due to the presence of a reversing patent ductus arteriosus; therefore, contrast medium remains in the ascending aorta for quite some time as demonstrated in Case II (Fig. 4 *B*). Little attention has been given to this pathognomonic angiographic sign.

Faint filling of an enlarged pulmonary artery through a large patent ductus arteriosus without significant flow of contrast medium into the pulmonary arterial bed is diagnostic of a reversing patent ductus arteriosus invariably found in this condition.

Most characteristically, however, the ascending aorta is found to be a small "string" giving rise to the coronary arteries which are usually densely filled. Due to the small size of the ascending aorta, it may be mistaken for the mammary or bronchial artery. This characteristic anatomic feature was missing in our 2 reported cases. Since, during the cardiac development, the size of blood vessels and cardiac chambers is largely governed by blood flow, it may be postulated in these 2 cases that aortic atresia occurred relatively late *in utero*. The recognition of this syndrome which may closely resemble extreme aortic valvular stenosis is of practical significance in view of the fact that such patients are potentially amenable to aortic valvulotomy.

SUMMARY AND CONCLUSION

Aortic atresia, the most common cause of cardiac death during the first week of life, is commonly diagnosed preoperatively by the physical, electrocardiographic and roentgenographic findings, which are briefly discussed. Contrary to previous reports, it was found that the size of the ascending aorta may be normal, as demonstrated by the 2 reported patients who came to postmortem examination.

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INTRAVENOUS ANGIOCARDIOGRAPHY, ABDOMINAL AORTOGRAPHY, AND PERIPHERAL ARTERIOGRAPHY WITH SINGLE ARM PRESSURE INJECTION*

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ADVANCES in the design and manufacture of electronic equipment for injection of concentrated opaque media and for rapid serial roentgenography have resulted in efficient, albeit expensive, apparatus. All sorts of automatic injectors and rapid serial film transport devices have found their way into the roentgen departments of the larger hospitals and medical centers. These have permitted alteration of manual techniques of injecting concentrated contrast materials with marked improvement in rapid serial contrast roentgenography of the cardiovascular system.

The practical method of visualizing the four cardiac chambers and the great blood vessels in man was reported by Robb and Steinberg[12] in 1938 and in this JOURNAL in January, 1939.[13] Angiocardiography was achieved by the rapid intravenous injection of a concentrated organic iodine compound. Speed of injection was secured by designing a special 12 gauge bore syringe and needle stop-cock unit which permitted the injection of 50 ml. of concentrated contrast agent in 1½ seconds. Speed of injection was also facilitated by elevation of the arm and instructing the patient to inhale and arrest breathing without performing the Valsalva maneuver.[14] The intravenous method was modified in 1959 in order to increase the bolus effect and to visualize the abdominal aorta and peripheral vascular system more regularly. Bilateral, simultaneous, intravenous injections enhanced angiocardiography,[18] achieved abdominal aortography and peripheral arteriography[16,17,21] and made it possible to inject larger quantities of contrast material (up to 100 ml.) for the cardiovascular visualization of heavily built and obese people, and patients with cardiomegaly owing to heart failure, valvular heart disease, or aortic aneurysms.[18]

Recently, 2 papers describing a single intravenous injection through the Robb-Steinberg needle for abdominal aortography have appeared in the literature. Cusmano and Gallagher[3] were the first to use a manually operated mechanical injector, while Hemley and Finby[10] later attached a Gidlund injector. Kanick and Finby[11] have also illustrated their recent article with angiocardiograms made following a single intravenous arm injection of angio-conray, using the Gidlund pressure apparatus attached to the Robb-Steinberg cannula. Following these reports, 158 patients at this center have had angiocardiographic, abdominal aortic, and peripheral vascular arteriographic studies after a single intravenous pressure injection of concentrated contrast material into an arm. In this article, the technique of single arm intravenous pressure injection using the Robb-Steinberg cannula for angiocardiography, abdominal aortography, and peripheral arteriography is illustrated, and the advantages over the simultaneous bilateral intravenous arm injections are discussed.

METHOD

Percutaneous insertion of the Robb-Steinberg needle stop-cock unit (Fig. 1*A*) is made with the patient in the supine posi-

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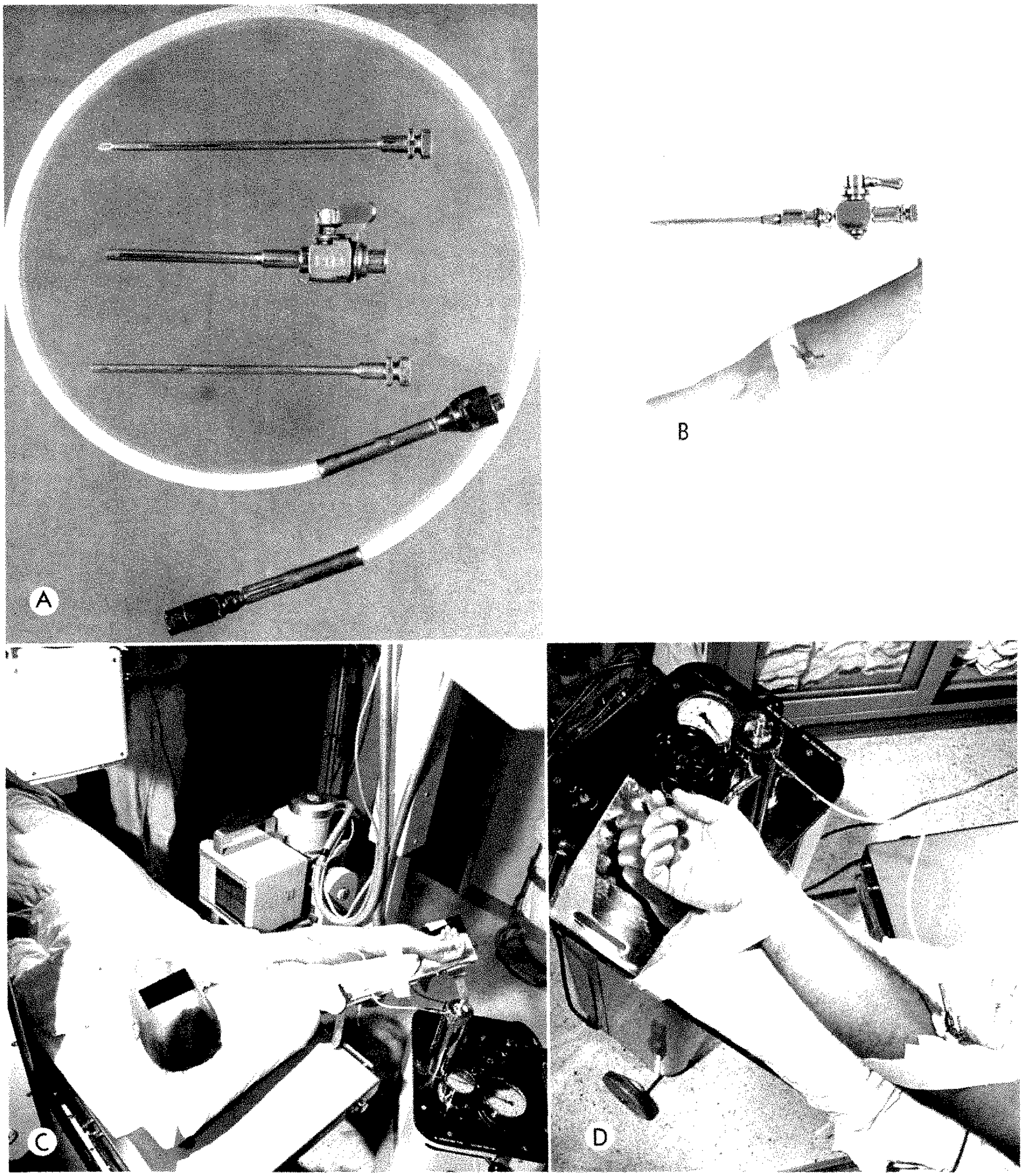

FIG. 1. *Technique of single arm pressure injection of contrast medium for intravenous angiocardiography, abdominal aortography, and peripheral arteriography.* (*A*) The unassembled Robb-Steinberg needle stop-cock unit (manufactured by Becton-Dickenson Co., Rutherford, N. J.) and the Piling wide bore connecting polyethylene adaptor tubing. (*B*) The assembled Robb-Steinberg cannula (upper) and its position in the arm, fixed with adhesive tape, after percutaneous insertion (lower). (*C*) Gidlund pressure injector connected to the Robb-Steinberg cannula. Note the elevated and raised position of the outstretched arm secured with adhesive strapping. (*D*) Close-up view of the Piling connecting tubing to the Robb-Steinberg cannula and Gidlund injector.

tion as previously described.[4,13,14] In the absence of a large arm vein, it is desirable to perform a cut-down for placement of the cannula. After the needle stop-cock unit is inserted into the vein, it is fixed in position with adhesive tape (Fig. 1*B*). The intravenous location of the needle is assured by flushing with normal physiologic saline solution. With arm elevated, the modified circulation time with sodium dehydrocholate (decholin) is determined. This is especially important when the abdominal aorta is to be visualized, and when there is heart disease (valvular and congestive heart failure). It is essential to explain the object of the circulation time and rehearse the procedure with the patient so that he will recognize the bitter taste of decholin and avoid the Valsalva maneuver. It is for this reason and in order to have the patient alert and cooperative that premedication with sedatives is not advised.

The details of the circulation time determination have been previously published.[16,17] However, it bears repetition because improper technique of circulation timing is the commonest reason for failure to visualize the abdominal aorta. Three milliliters of a 20 per cent solution of decholin are mixed with 15 ml. of physiologic saline solution in a 20 ml. syringe and attached to the Robb-Steinberg cannula. The arm is elevated making certain that it is relaxed at the shoulder (this avoids physiologic obstruction of the subclavian vein underneath the first rib). The stop-cock is opened and the patient is instructed to "breathe out" gently. Then he is asked to "breathe in" making certain that only a moderate breath is taken, for it is important to avoid the Valsalva maneuver, which tends to delay circulation into the heart. Injection of the decholin-saline mixture is made simultaneously with the direction to "breathe in." This is also the signal for the technician to start the stop watch. Once the injection is completed, the patient, having been told to anticipate a bitter taste, is urged to go through the motion of tasting by smacking his lips, tongue, and mouth; this automatically releases the tendency to perform the Valsalva maneuver. The onset of the bitter taste is recorded by stop watch. The modified circulation time from arm-to-tongue is a valuable guide for the duration of the roentgen exposure for cardiovascular angiography, and for abdominal aortography and peripheral arteriography.[16,17] If the accuracy of this determination is uncertain, it is essential to repeat the procedure.

The position of the patient for single arm pressure injection is supine on a horizontally placed rapid, multiple, serial 14 by 14 inch Schönander magazine (Fig. 1*C*). The outstretched extended arm containing a Robb-Steinberg needle taped with adhesive is connected to the Gidlund pressure injector by a special (Piling 13 gauge) polyethylene adapter connector (Fig. 1*C*). The details of the connection between the stop-cock unit of the Robb-Steinberg needle and the Gidlund pressure apparatus are shown in Figure 1*D*. Additional adhesive taping to fix the adapter connecting tubing can be used to stabilize and prevent dislocation of the Robb-Steinberg cannula.

Injection of the concentrated contrast medium, usually angio-conray,[11,19] is made in $1\frac{1}{2}$ to 2 seconds at a pressure of 5 to 6 kg./cm.^{2}. The dose of contrast material is 1 ml./kg. of body weight. Before injection of contrast material, a small quantity, usually 1 to 2 ml., is manually injected by turning the wheel of the Gidlund injector. This is done to make certain that the cannula is well within the vein. In the absence of arm pain, tissue infiltration is ruled out and the angiographic studies are begun. For angiocardiography, filming begins at the precise moment of injection and continues for the duration of the circulation time. The patient is also forewarned and reassured about an intense wave of heat and throbbing in the head of short duration. Programming, depending on the number of films per second desired with delays at appropriate intervals, may be estimated with great accuracy once the circulation time has been determined. For instance, in rheumatic heart disease every other slot of the Schönander magazine is loaded for 15 films and this provides a study of either 5 or 22 seconds duration, depending on whether roentgen exposures are made at 1 second or 1.5 second intervals. Should the circulation time be longer than 22 seconds, predetermined delays during the pulmonary circulatory (arteriovenous) phases may be secured by programming with the Schönander electronic timer device.

Abdominal aortograms and peripheral arteriograms are obtained by utilizing an automatic movable table (the Koordinate) over a Schönander magazine.[20] Here too, the circulation time is of paramount importance. Usually, filming is begun 1 second before the decholin circulation time. Five roentgenograms at intervals of 0.75 second are made. To prevent blurring during movement of the table, the sixth slot in the Schönander magazine is not loaded. Exposures of the pelvis (in the second table stop) are then made at intervals of 0.75 second for 3 films. Similarly, 3 exposures of the thighs and legs are made, respectively, following a blank slot at the end of 3 sets of films.

Since the patient is supine and stationary during angiocardiography, the Piling polyethylene connecting adapter tubing between the Gidlund syringe and needle stopcock unit need not be disconnected. Because of automatic movements of the table during peripheral arteriography,[20] it is necessary to disconnect one end of the connecting Piling tubing from either the patient or the Gidlund injector. After practice, this can be readily achieved before roentgenography of the abdominal aorta begins. At the completion of the studies and while the films are being processed, the solid hub bore (Fig. 1*A*) is inserted into the lumen of the Robb-Steinberg needle to prevent clotting in the cannula. In aortotomography,[21] however, detachment of the Gidlund injection device from the Piling connecting tubing is not necessary until completion of the studies since the patient remains in a stationary position during the multiple filming. Removal of the needle is followed by tight bandaging of the needle puncture site with sterile gauze. Usually, the snug dressing can be removed after 2 hours, and a light sterile band-aid applied.

RESULTS

Illustrative examples of the results obtained by single arm pressure injection of concentrated agents in the first 158 patients are shown in Figures 2 through 6, inclusive. Excellent visualization of the cardiovascular system was secured in heavily built and obese patients with aortic aneurysms (Fig. 2, *D*, *E*, *F*, *G*, and *H;* and 3, *C*, *D*, *E* and *F*). Indeed, the patient shown in Figure 3 also had congestive heart failure with prolongation of the circulation time and dilated cardiac chambers owing to aortic regurgitation, and yet, excellent opacification of all the left heart structures was achieved (Fig. 3, *D* and *F*). A patient with a ventricular septal aneurysm following myocardial infarction due to arteriosclerotic heart disease is shown in Figure 4, *A* and *B*. With this technique the abdominal aorta and peripheral vascular system of a woman with severe intermittent claudication of both hips and legs and with abdominal bruits were well visualized. Extensive arteriosclerotic thrombotic occlusive and aneurysmal disease of the abdominal aorta was revealed (Fig. 5*A*). Intact aortoiliac arteries were demonstrated (Fig. 5, *B* and *C*) and the run-off below the knees was excellent (Fig. 5*D*)—features which made her an ideal operative case.

Finally, in a miscellaneous group of patients, excellent opacification of the cardiovascular, abdominal aortic, and peripheral vascular systems was obtained (Fig. 6, *A*–*D*). Figure 6*A* shows severe buckling of the lower portion of the thoracic aorta in a 72 year old woman who was admitted in shock. Because of severe chest pain and an unusual bulge in the left hilus region, she was suspected of having dissection of the thoracic aorta. Another patient with a large abdominal aortic aneurysm (Fig. 6*B*) also had intact aortoiliac arterial vessels with good run-off, and so resection of the

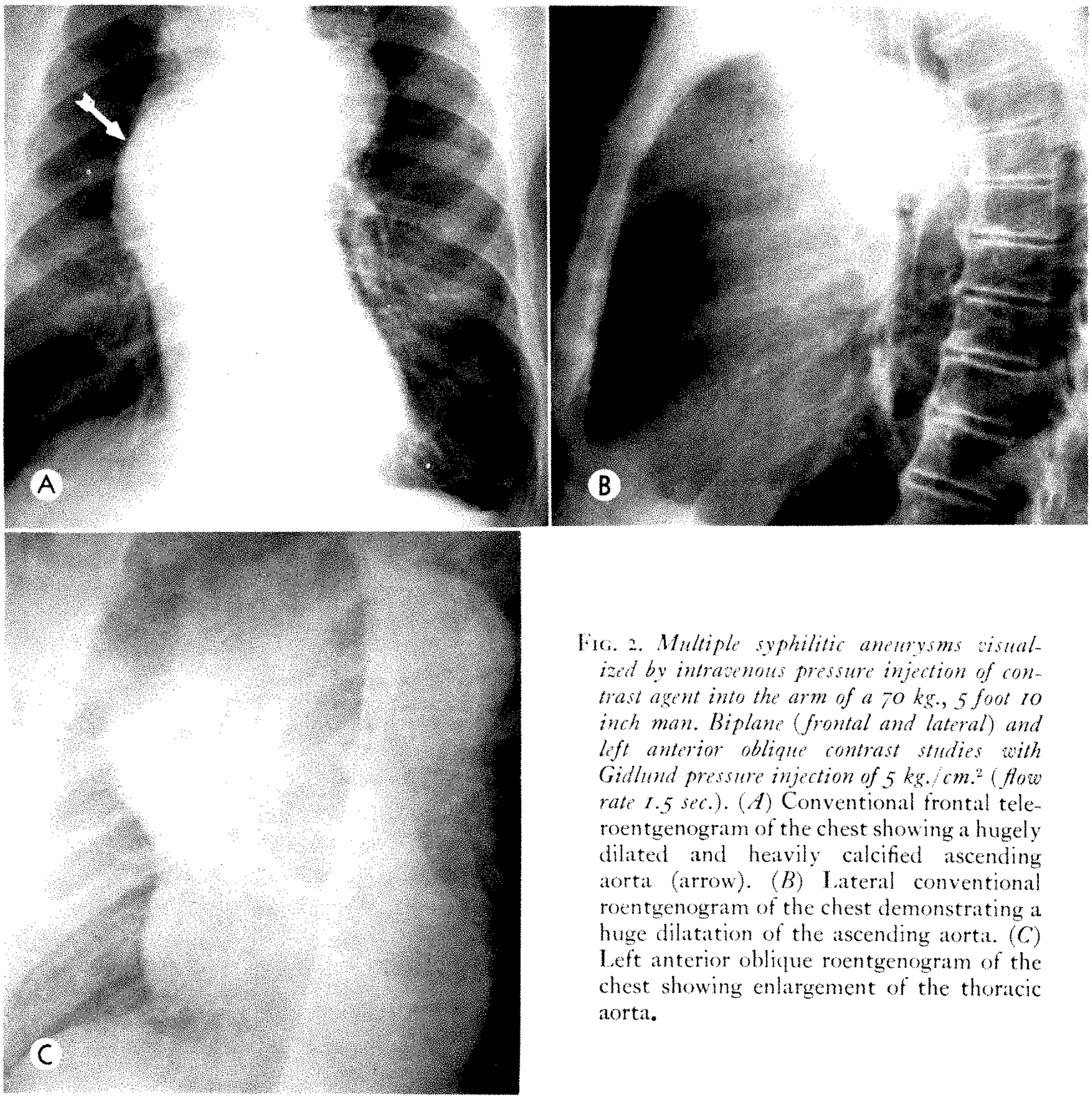

Fig. 2. *Multiple syphilitic aneurysms visualized by intravenous pressure injection of contrast agent into the arm of a 70 kg., 5 foot 10 inch man. Biplane (frontal and lateral) and left anterior oblique contrast studies with Gidlund pressure injection of 5 kg./cm.^{2} (flow rate 1.5 sec.).* (*A*) Conventional frontal teleroentgenogram of the chest showing a hugely dilated and heavily calcified ascending aorta (arrow). (*B*) Lateral conventional roentgenogram of the chest demonstrating a huge dilatation of the ascending aorta. (*C*) Left anterior oblique roentgenogram of the chest showing enlargement of the thoracic aorta.

aneurysm was feasible. An aortoiliac teflon graft was visualized 1 year after excision of an abdominal aortic aneurysm (Fig. 6*C*). Finally, occlusion of the left popliteal artery in a 95 year old woman, the oldest person in the series, with intermittent claudication of the left leg was demonstrated (Fig. 6*D*).

DISCUSSION

The advantages of a single arm over the double arm injection technique for angiocardiography, abdominal aortography, and peripheral arteriography are evident and were recently succinctly summarized by Cusmano and Gallagher[3] as follows: Only one arm is injected and, therefore, there is less discomfort to the patient; less time is required to insert a single needle; and finally one less radiologist is involved in the procedure. However, the automatic nature of the injection does not permit discontinuance of the injection when there is infiltration of the contrast material into the arm tissues. On the other hand, when infiltration occurs during manual injection of contrast material with the Robb-Steinberg syringe, local infiltration can usually be im-

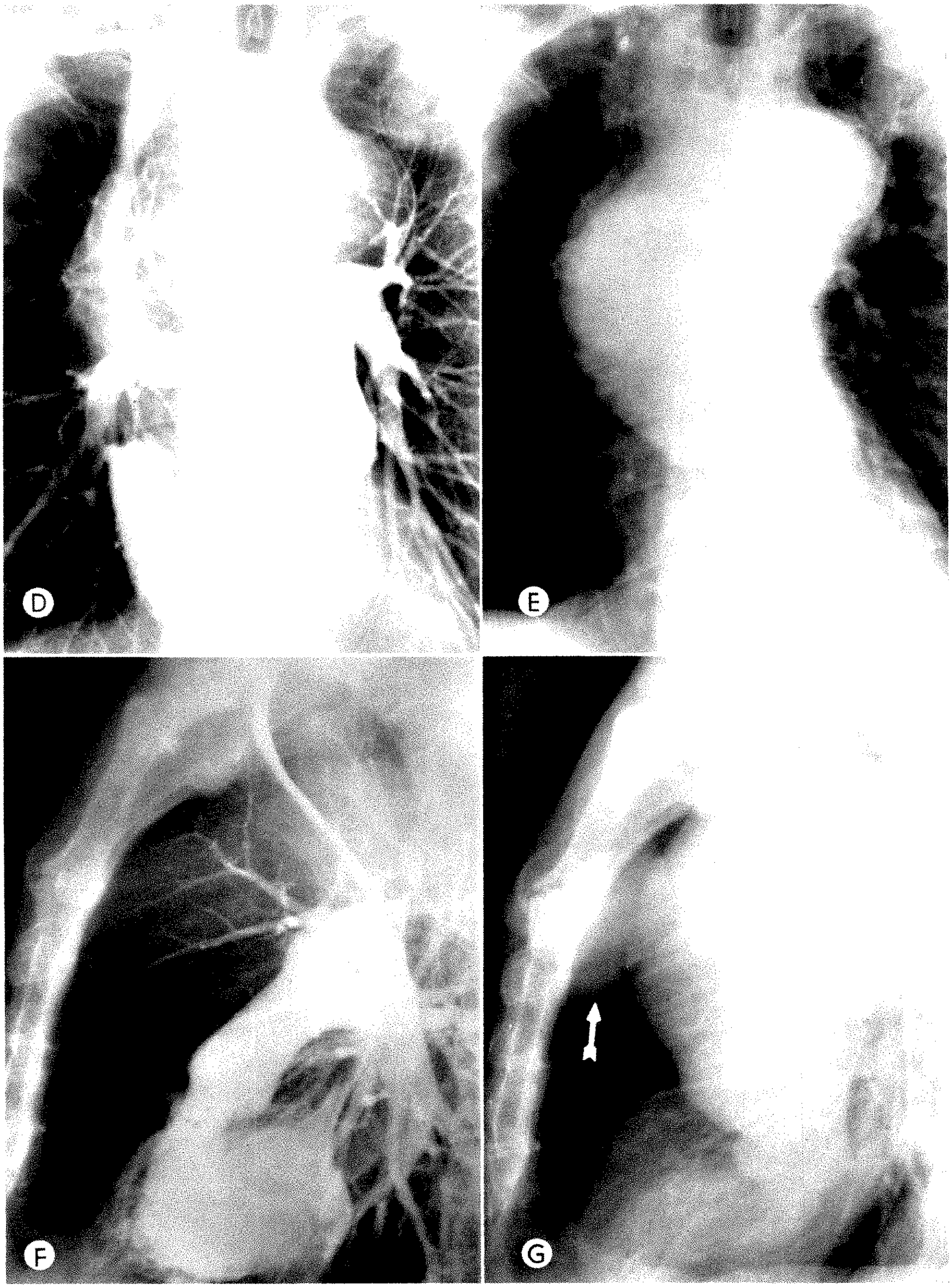

Fig. 2. (*D*) Frontal angiocardiogram showing the displaced superior vena cava and pulmonary artery. (*E*) Frontal angiocardiogram demonstrating the huge fusiform aneurysm of the ascending aorta. (*F*) Biplane, lateral angiocardiogram of *D* showing the displaced superior vena cava and pulmonary artery. (*G*) Biplane, lateral angiocardiogram of *E* showing the huge fusiform aneurysm of the ascending aorta with dilatation of the innominate artery. Note also the saccular aneurysm arising from the mid-ascending aortic aneurysm (arrow).

mediately recognized and the injection stopped, thereby preventing injection of large quantities of the contrast medium.

Infiltration with contrast material occurred only once early in the experience with use of pressure for arm injection. To decrease the risk of arm infiltration, it is important to make certain that the cannula is well placed in the vein. This can easily and quickly be tested by injecting physiologic saline solution through the cannula and watching for extravasation. Reinsertion of the Robb-Steinberg needle into another vein either percutaneously or by cut-down is recommended whenever there is local edema of the arm following physiologic saline injection. Finally, as a further precaution, just before injection of the contrast medium a small test dose of 1–2 ml. of the contrast agent should be injected with the Gidlund apparatus as described above. As an added safety measure in order to prevent dislodgement of the needle during injection, the Piling connecting tubing is immobilized by adhesive tape for angiocardiography or may be held for the duration of the injection (2 seconds) during abdominal aortography and peripheral arteriography. At the completion of the injection, the tubing can be disconnected from the Gidlund apparatus so that movement of the automatic table will be unhindered. In any event, it is recommended that both the needle and Piling connector tubing be well immobilized during the injection (Fig. 1, *C* and *D*).

Local infiltration of the arm tissues with concentrated contrast agents, even in small quantities, is extremely painful. Immediate treatment by injection of large quantities of physiologic saline solution into the dislodged cannula will considerably dilute the concentration of the contrast agent, especially if the cannula is no longer in the vein, and will reduce pain and tissue damage. Hyaluronidase solution, because it hastens absorption of contrast media, is also injected into the tissues via the dislodged cannula. If pain persists, injection of procaine solution, 1 per cent, locally into the painful areas is advised, and this medication should be continued until pain is alleviated. Sedation with barbiturates or narcotics may also be necessary. Hot packs applied to the site of infiltration will also give comfort. Localized slough of the skin has been rare following the procedures outlined above.

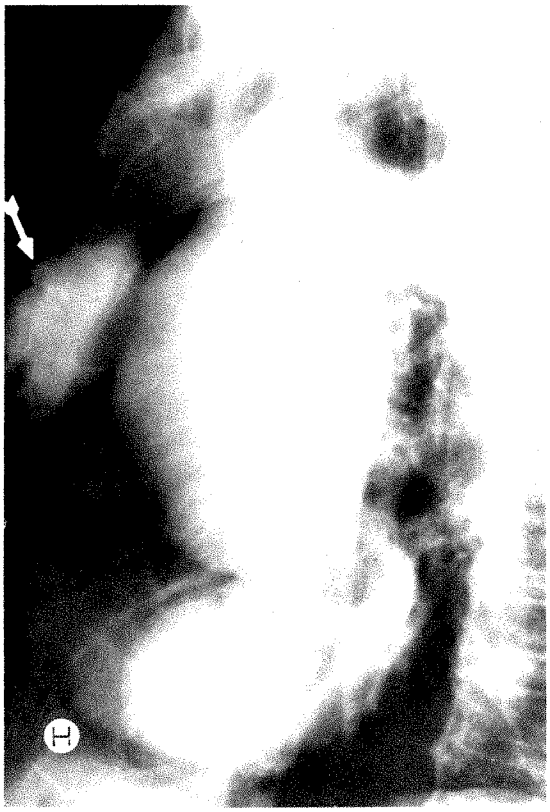

Fig. 2. (*H*) Left anterior oblique angiocardiogram also demonstrating the massive aneurysm of the ascending aorta with the anterolateral saccular aneurysm (arrow).

Insertion of catheters into the arm and superior vena cava has also been used for intravenous angiocardiography and abdominal aortography and is apparently very satisfactory.[1,2,5,7,9,15,23] Usually, this requires a cut-down, although the percutaneous technique for insertion of large catheters is available. In any event, large bore catheters of at least 12 gauge are essential. Although catheters rather than large bore cannulas are recommended by some because they prevent stagnation of con-

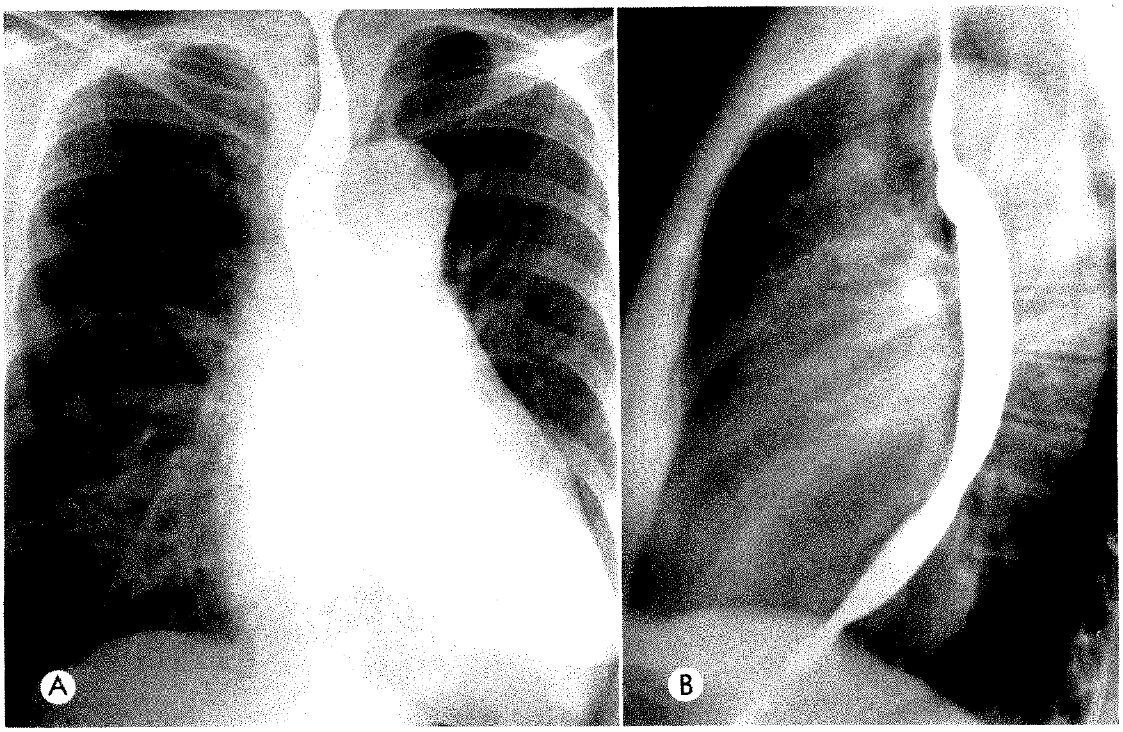

Fig. 3. *Aneurysmal dilatation of the ascending aorta and aortic sinuses (of Valsalva) visualized by single arm intravenous pressure injection of contrast medium in a 56 year old tall woman weighing 65 kg.* (*A*) Frontal esophagram showing a large heart, prominent ascending aorta, and aortic knob. (*B*) Lateral esophagram demonstrating the dilated ascending aorta with posterior displacement of the esophagus.

trast material in the arms, trapping of contrast material in the arm or neck veins can be avoided if the arm used for injection is elevated and the patient is taught to avoid the Valsalva maneuver. Indeed, when the Valsalva maneuver was inadvertently performed during an angiocardiographic study made recently at another hospital, marked regurgitation of contrast material into the jugular veins occurred even though the catheter had been inserted into the right atrium.

Pressures of 5 to 6 kg./cm.^{2} with the Gidlund apparatus have uniformly resulted in flow rates of 1½ to 2 seconds when angioconray (iothalamic acid, 80 per cent) was used. Flow rates of the more viscid concentrated organic iodide contrast agents have

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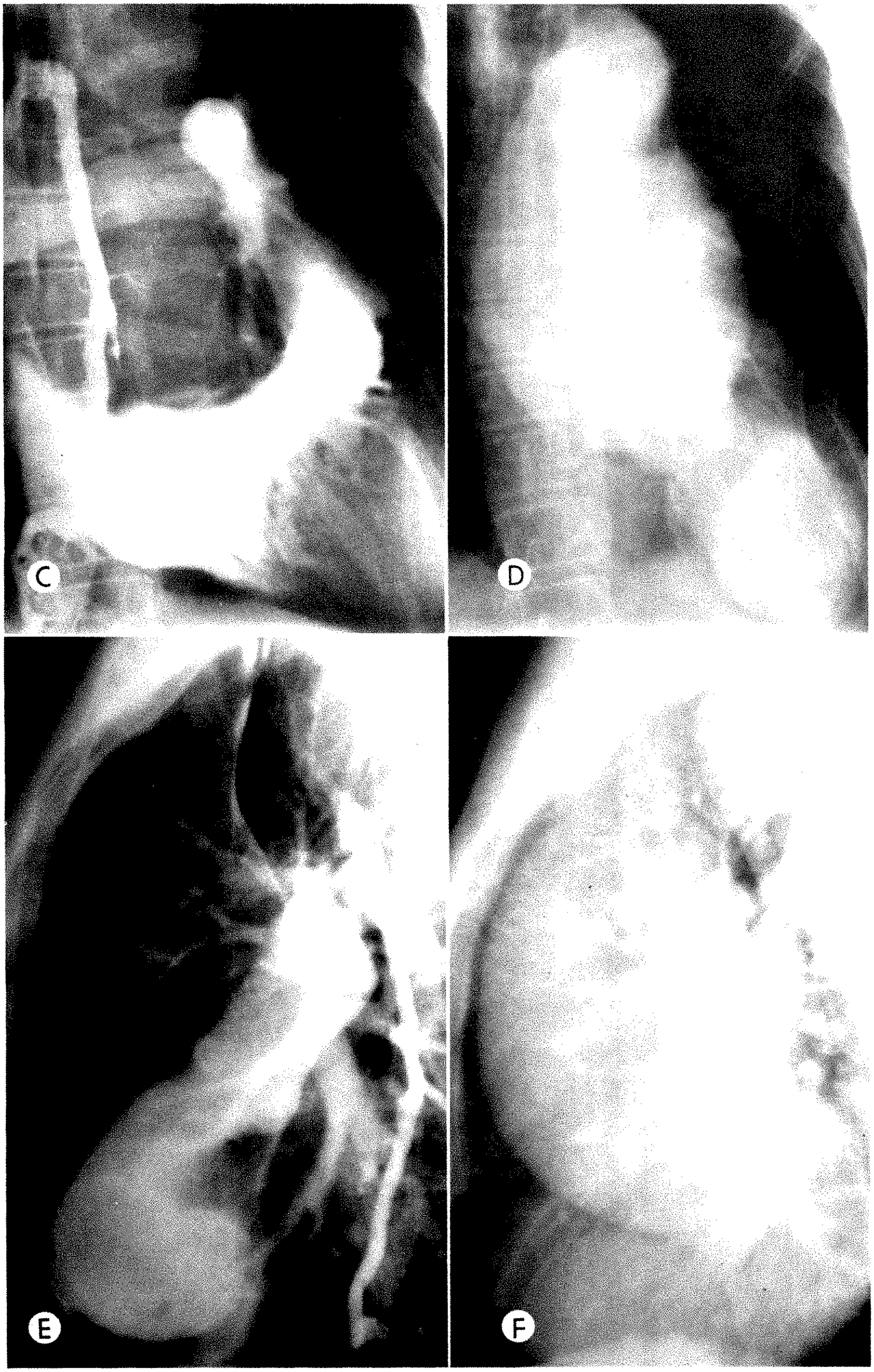
Fig. 3. (*C*) Frontal angiocardiogram revealing the displaced and flattened superior vena cava with reflux filling of the azygos system. Note the huge filling defect with displacement of the right atrium and outflow tract of the right ventricle. (*D*) Frontal angiocardiogram showing the huge aneurysm of the ascending aorta and aortic sinuses (of Valsalva). (*E*) Biplane, lateral angiocardiogram of *C* showing the dilatation of the superior vena cava and retrograde opacification of the azygos system. Note the dilated pulmonary artery. (*F*) Biplane lateral angiocardiogram of *D* again revealing the aneurysm of the ascending aorta and aortic sinuses.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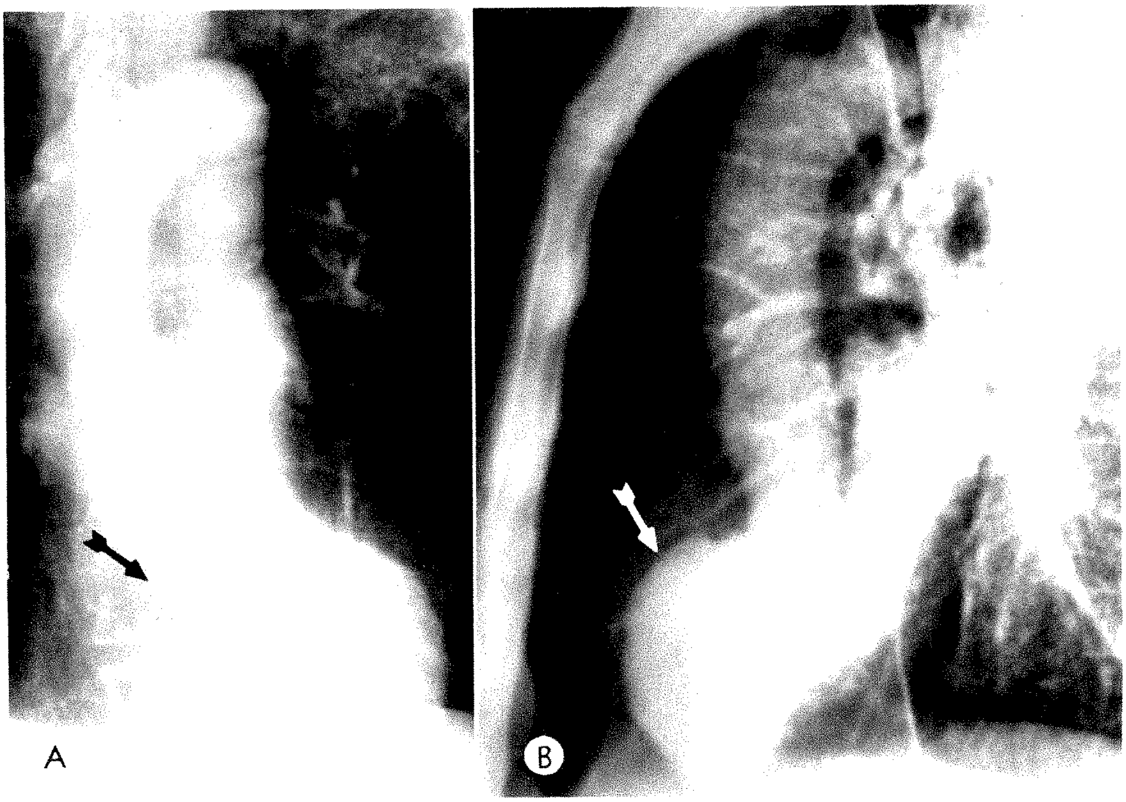

Fig. 4. *Ventricular septal aneurysm opacified by a single arm pressure injection of organic iodide into a 62 year old man following recent myocardial infarction.* (*A*) Frontal angiocardiogram revealing an aneurysm (arrow) of the ventricular septum. (*B*) Biplane lateral angiocardiogram of *A* showing the aneurysm of the ventricular septum to better advantage.

been slower than with angio-conray; therefore, the use of angio-conray[11,19] assures a speed of injection between 1½ to 2 seconds, a rate which has long been found satisfactory for intravenous angiocardiography during the past 25 years.[4,14,16] Other, less expensive pressure devices, whether they release pressure automatically or after manual manipulation, are suitable providing they permit a flow rate of 1½ to 2 seconds.

Failure to visualize the abdominal aorta, irrespective to whether double or single arm injections are made, is most often caused by imperfect circulation time determination. Since the determination of the decholin circulation time is of great importance, care is necessary that a large bore 12 gauge cannula such as the Robb-Steinberg needle is inserted into the arm. The arm must be elevated and the patient placed in the position that he will assume for contrast cardiovascular visualization studies. The patient must also be alerted to anticipate the bitter taste and instructed how to avoid the Valsalva maneuver. Indeed, it is because the patient's cooperation is essential for the securing of the correct circulation time and later the contrast studies, that premedication of any type is avoided. Furthermore, an accurate stop watch, and not the ordinary wrist watch, must be used for recording the moment of bitter taste. Finally, roentgen apparatus, especially serial automatic electronic devices, often have varying lags before roentgen exposure and initiation of the movement of the serial film devices. These lags must be ascertained and allowances made for them. Only then will the circulation time be meaningful.

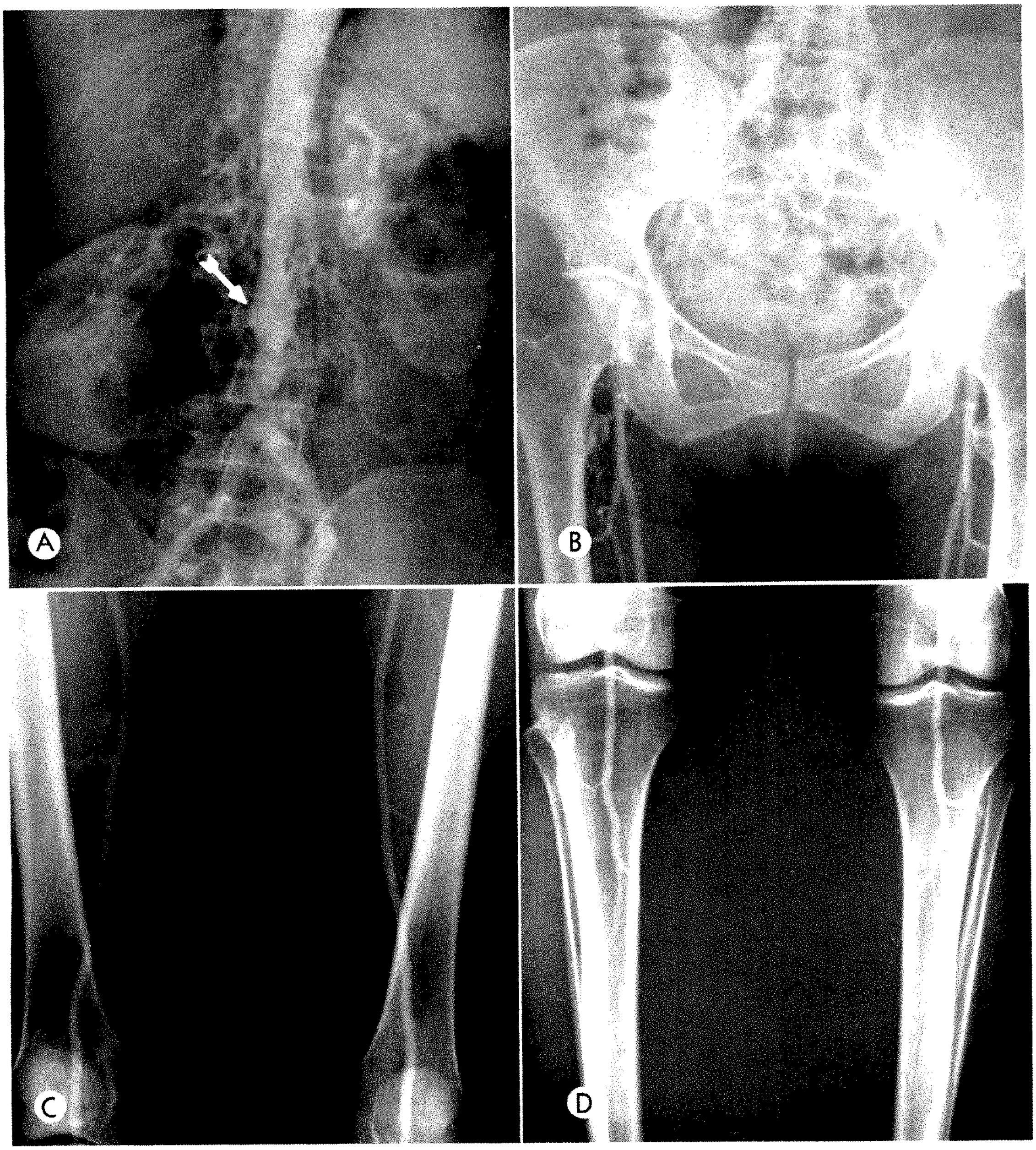

Fig. 5. *Severe arteriosclerotic occlusive and aneurysmal disease of the abdominal aorta visualized with a single arm pressure injection of contrast material in a 57 year old woman studied with the automatic movable table.*[20] (*A*) Intravenous abdominal aortogram disclosing the markedly irregular and narrowed abdominal aorta. Note a small saccular aneurysm (arrow). (*B*) The aortoiliac system is tortuous, but intact. (*C*) The superficial femoral arteries are also intact. (*D*) The last of the serial roentgenograms shows excellent run-off below both knees.

SUMMARY AND CONCLUSIONS

A single arm pressure injection of concentrated contrast material for angiocardiography, abdominal aortography, and peripheral arteriography has recently displaced the bilateral simultaneous arm injection technique at The New York Hospital. The Robb-Steinberg (12 gauge) cannula is connected by a short polyethylene adapter tubing to the Gidlund injector.

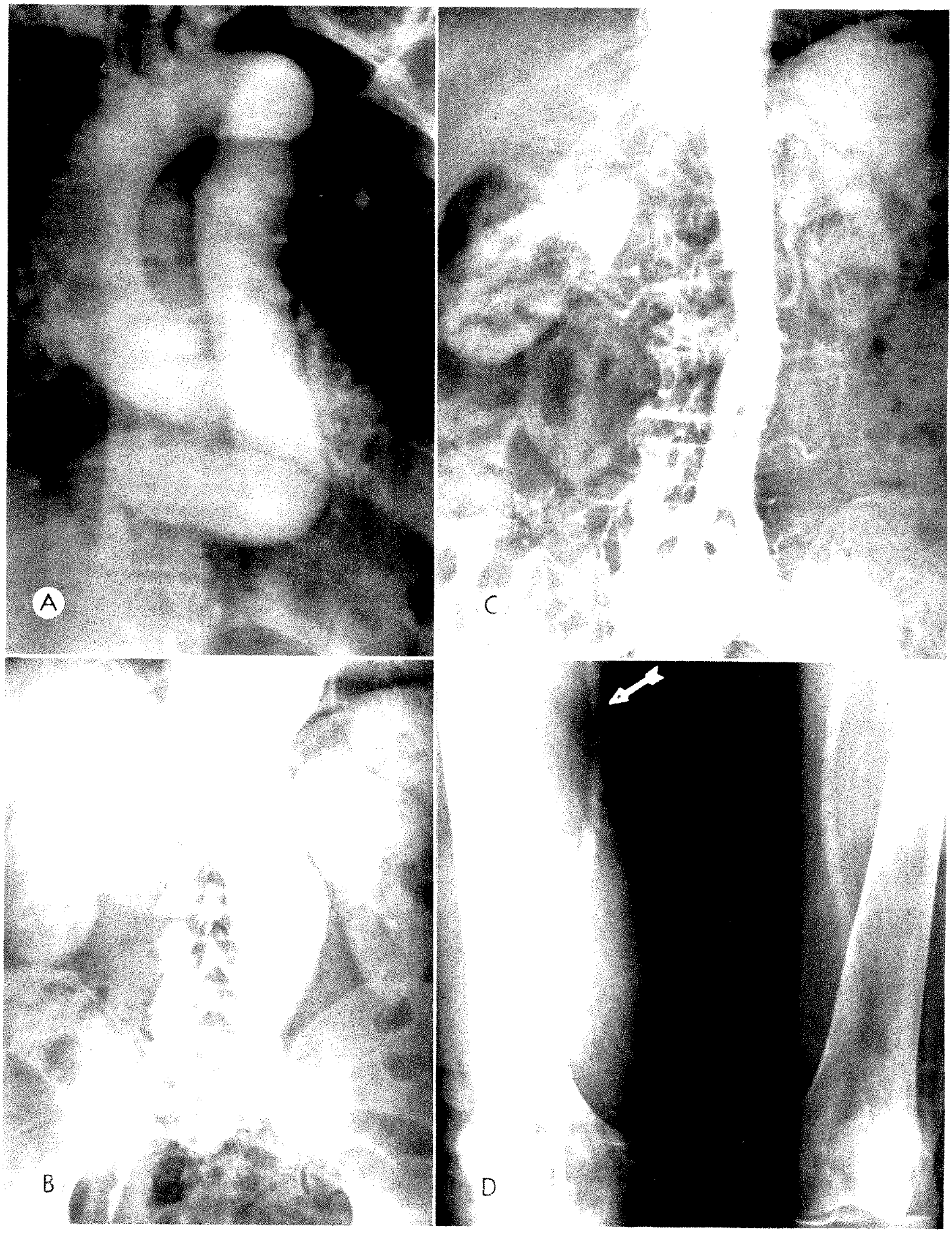

FIG. 6. *Multiple angiograms showing the results of the single arm pressure injector method in intravenous angiocardiography, abdominal aortography, and peripheral arteriography.* (*A*) Frontal angiocardiogram of a 72 year old woman, weighing 72 kg. and admitted in shock. A markedly elongated, unfolded, and tortuous thoracic aorta was demonstrated which ruled out dissecting aneurysm. (*B*) Abdominal aortic aneurysm, lined with calcifications in a thrombus, in a 63 year old man. (*C*) Teflon graft replacement of an abdominal aortic aneurysm, excised 1 year earlier in a 56 year old woman. (*D*) Peripheral arteriogram of a 95 year old woman showing occlusion of the superficial femoral artery at the left thigh. Note the soft tissue defect in the right upper thigh (arrow), the site of a previously excised malignant fibrosarcoma.

Angio-conray, 1 ml. for each kg. of body weight, is injected at pressures of 5–6 kg./cm.^{2}, which results in flows of $1\frac{1}{2}$ to 2 seconds duration. The arm is elevated during injection. The patient is taught to inspire at the onset of injection, and to avoid the Valsalva maneuver.

This technique has resulted in superb angiocardiograms, abdominal aortograms, and peripheral arteriograms even in muscular and overweight patients. Huge thoracic aortic and abdominal aortic aneurysms, and enlarged and dilated hearts of patients in heart failure have been regularly visualized.

The advantage of the single arm pressure technique over the double arm injections is that it avoids insertion of two needles, it makes participation of another physician unnecessary, and in the case of angiocardiography allows the injector to remain in the control room away from the radiation zone. The principal disadvantages are the greater expense of the automatic pressure injector apparatus and the danger of infiltration of the concentrated contrast agent owing to the automatic nature of the injection. The former can be remedied by using less expensive injectors, automatic and manual. The latter can be avoided by making certain that the needle is well placed within the vein by preliminary testing for infiltration by injection with physiologic saline solution. In case of doubt, reinsertion of the Robb-Steinberg cannula, percutaneously or by cut-down, is essential.

Improper determination of the circulation time with decholin is the commonest reason for failure to visualize the abdominal aorta and peripheral vascular circulation with the intravenous method. For successful performance of the circulation time—and contrast visualization of the abdominal aorta and peripheral vascular circulation—the following are essential: A large bore (12 gauge) Robb-Steinberg cannula or catheter must be well inserted into the largest vein of the arm. The patient must be taught to anticipate the bitter taste of decholin. He must be instructed how to breathe and how to avoid the Valsalva maneuver. A stop watch, rather than a wrist watch, must be used for timing. Lags inherent in roentgen apparatus, whether of exposure time or of serial roentgen electronic devices, must be ascertained, and allowance for these made. Finally, reassuring an alert nonsedated patient concerning the intense wave of heat will regularly result in successful angiographic studies.

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ADDENDUM

Since this paper was prepared, experiences with another route for visualization of the aorta were reported by Friedenberg, M. J., and Edelman, A. N. Percutaneous transfemoral right atrial aortography. *Radiology*, 1963, *81*, 584–588.

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ANGIOGRAPHIC ARTEFACTS WHICH SIMULATE OR MASK ABNORMALITY*

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ANGIOGRAPHIC procedures may in themselves create abnormalities and simulate disease in vessels. These techniques may also fail to show abnormalities because of a variety of artefacts. The following classification of angiographic artefacts is intended to help radiologists who are beginning to gain experience in this field. The classification is the result of a review of more than 1,200 vascular studies at the San Francisco Veterans Administration Hospital and at the University of California Hospitals, San Francisco, between November, 1962 and November, 1963.

Roentgenographic vascular artefacts may be the result of (1) instrumentation, (2) flow, (3) position, and (4) technique.

INSTRUMENTATION ARTEFACTS

Instrumentation causes a variety of artefacts that result from poor selection and control of catheter, extraluminal injection, and spasm. Under poor control of the catheter, contrast material may not be deposited where it is intended. The resultant reduced opacification can simulate or fail to show true abnormalities. Extraluminal filling may cover true abnormalities, or simulate arteriosclerosis or occlusion when none exists. Spasm may be misinterpreted as arteriosclerosis or block, and excess filling may be misconstrued as collateral branches.

SELECTION AND CONTROL OF CATHETER

Too small a catheter, unsuitable holes, or low pressure of injection may cause insufficient delivery. Although contrast medium is seen within the vessel in these conditions, it may not be sufficient to reveal disease (Fig. 1). Conversely, excessive filling in smaller vessels may simulate collateral branches and produce spasm that suggests block. Displacement of the catheter tip by the jet effect of injection may produce failure of filling in the desired area, or filling in an undesired area (Fig. 2). The catheter frequently returns to its original position after the reaction of injection so that the incorrect injection site may not be recognized. Whip of the catheter into a small vessel may produce overloading, damage to the organ supplied, blowout, and spasm.

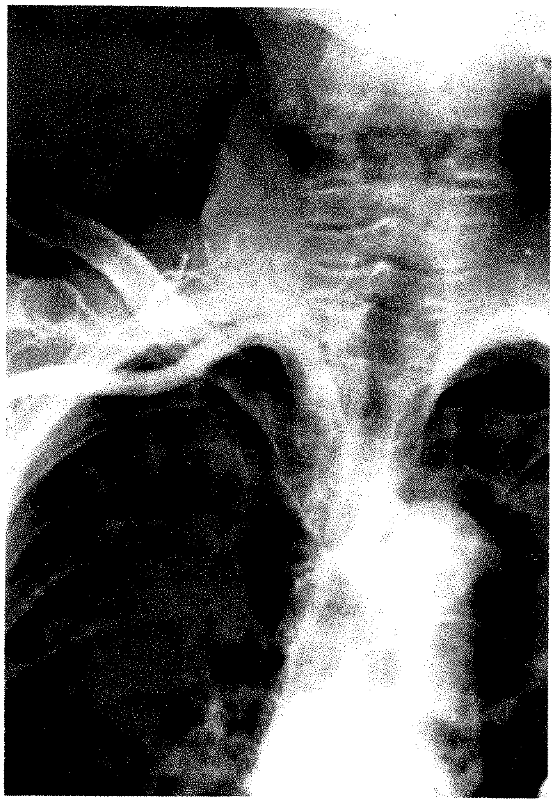

Fig. 1. Low pressure and short catheter result in failure of filling of proximal right subclavian artery, simulating occlusion.

When the catheter is inserted for a given length without roentgen control, the cathe-

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* From the Department of Radiology, University of California School of Medicine, San Francisco, California.

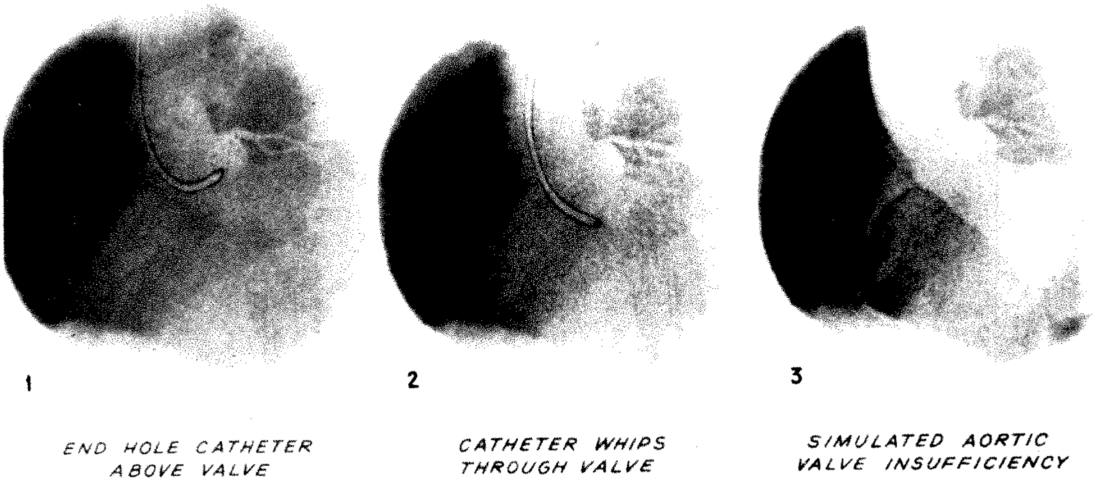
1
2
3
END HOLE CATHETER ABOVE VALVE
CATHETER WHIPS THROUGH VALVE
SIMULATED AORTIC VALVE INSUFFICIENCY

Fig. 2. Retouched frames selected from a 16 mm. cineangiogram. Aortic insufficiency is simulated when catheter slips through aortic valve from the jet effect of injection.

ter tip may terminate above or below the area of interest. The contrast medium may then not demonstrate the appropriate parts. The catheter may pass into the wrong vessel (Fig. 3) or turn into the wrong direction. Without the use of fluoroscopy, the catheter may pass even through the vessel wall with little response from the patient to indicate trouble (Fig. 4). Adequate fluoroscopic control, or, less preferably, roentgenographic control of insertion of catheter, aids in the reduction of artefacts of this procedure.

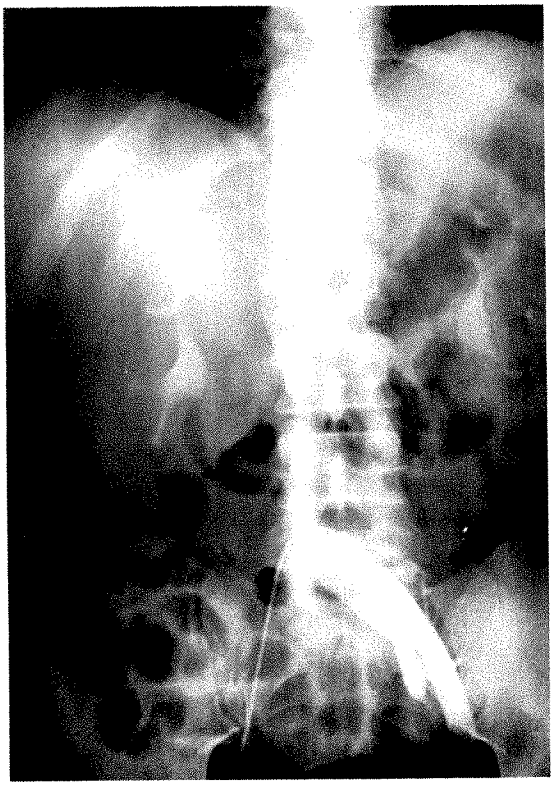

Fig. 3. Attempted catheterization of right atrium via right femoral vein approach. Catheter passed into left common iliac vein.

EXTRALUMINAL INJECTION

Many authors have described the problems of extraluminal injections of contrast material.[5,7,8,11-13,15] Extraluminal filling often occurs simultaneously with intraluminal injection of contrast material when the catheter or needle hole is partially in the lumen and in the wall of the vessel. The actual pathologic changes in anatomy and point of dissection produced by an intramural injection may not be certain in each case. The roentgen manifestations can be classified as subadventitial, subintimal, extravascular, or intraclot.

Contrast material deposited subadventitially or perivascularly beneath the vascular sheath may simulate intraluminal filling so that recognition of this artefact, without serial angiography, sometimes is difficult. Vascular occlusion can be simulated where subadventitial dissection ends (Fig. 5, *A* and *B*).

Subintimally placed contrast material may cause arterial narrowing or complete occlusion. By tearing the intima, a subintimal flap may be formed which retains the opaque medium and obstructs the vessels.[15] A subintimal hematoma will cause a negative filling defect in a roentgenogram of the contrast column and may also obstruct the vessel. When lumen and wall of a vessel receive a part of the injection simultaneously, early serial roentgenograms may show only subadventitial dissection. The subintimal hematoma may increase in size later and be seen only on delayed studies (Fig. 5*C*). A subintimal filling defect may be unrecognized on trial roentgenograms of a small injection. A full delivery of medium following failure to recognize intramural placement on a test injection may completely block the blood supply to vital organs (Fig. 6, *A*, *B* and *C*). The obstruction from intramural injections clears spontaneously in a few days or, sometimes, in a few hours, and significant sequelae have been infrequent in our experience (Fig. 5, *D* and *E*).

Intramural injection may be suspected when a delayed roentgenogram shows pooled contrast material remaining in the subadventitial space. When injected saline shows diluted blood upon withdrawal, incorrect extraluminal placement of the needle may be surmised. This sometimes helpful test was first described by Newton and Couch.[8] Sometimes occlusion resulting from partial intramural injection and obscured by surrounding contrast medium is indicated by reversal in flow of intraluminal contrast material.

Passage of the catheter into intraluminal clots in an aneurysm may simulate extravascular placement. What appears as extravascular passage of a catheter out of aortic or iliac arteries more often is passage into grumous material within an aneurysm. Both types have been seen here and were proved surgically.

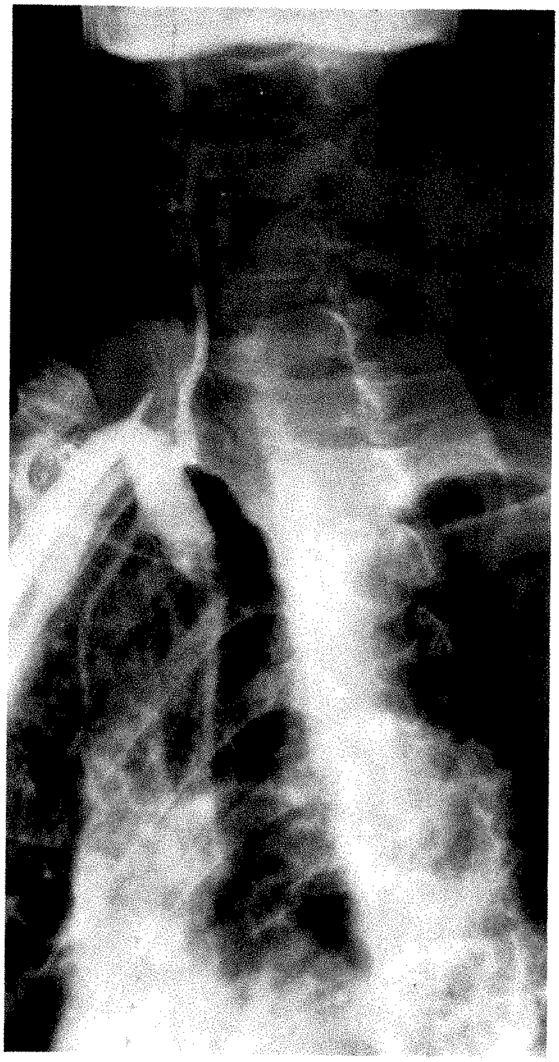

FIG. 4. Right subclavian arteriogram. Tip of catheter passed through the vessel wall but plugging of the end-hole prevented extravasation.

ARTERIAL SPASM

Spasm may simulate arteriosclerosis, vascular occlusion (Fig. 7, *A* and *B*), or fibromuscular hyperplasia.[10] In larger vessels, a crimped, accordion-pleated appearance of spasm may simulate a dacron graft. Although this spasm somewhat resembles that in peripheral vessels of Reynaud's disease, a relationship to this phenomenon in these patients has not been investigated by us.

Intraluminal foreign bodies, a sharp rise in the intravascular pressure, or irritation

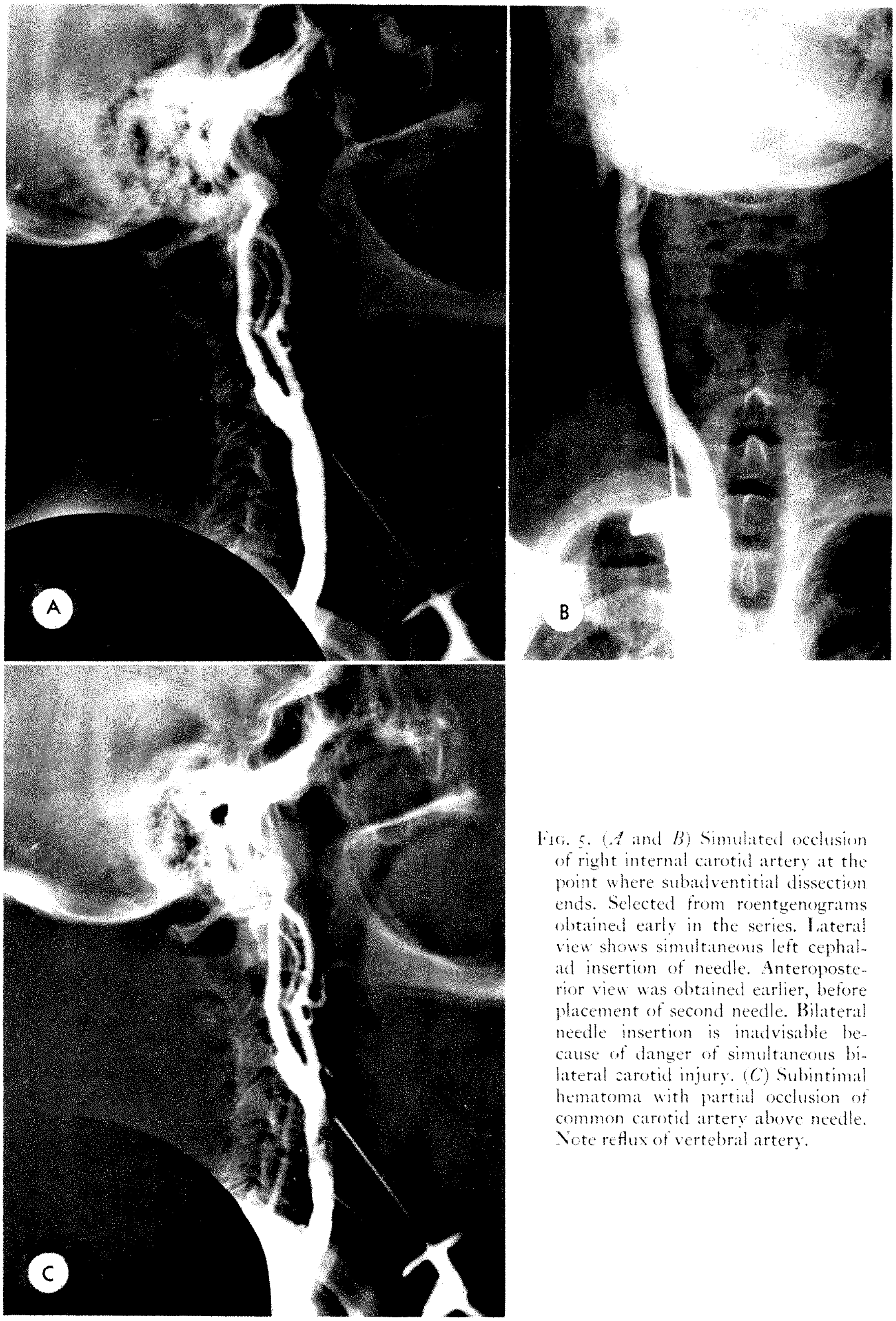

FIG. 5. (*A* and *B*) Simulated occlusion of right internal carotid artery at the point where subadventitial dissection ends. Selected from roentgenograms obtained early in the series. Lateral view shows simultaneous left cephalad insertion of needle. Anteroposterior view was obtained earlier, before placement of second needle. Bilateral needle insertion is inadvisable because of danger of simultaneous bilateral carotid injury. (*C*) Subintimal hematoma with partial occlusion of common carotid artery above needle. Note reflux of vertebral artery.

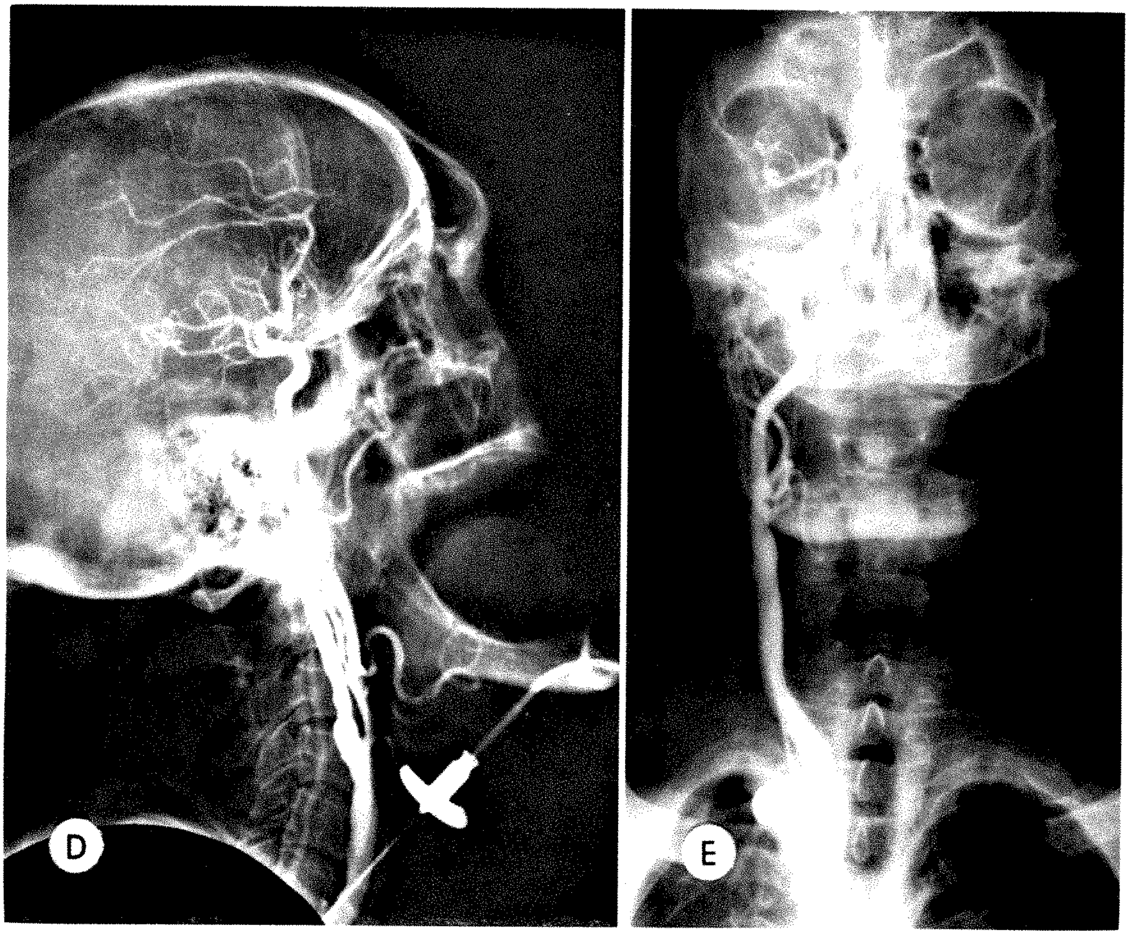

Fig. 5. (*D* and *E*) Same patient re-examined 1 week later at which time occlusion was not seen. Separate injections. The faint filling of the vertebral artery seen in the lateral view is the result of reflux into the subclavian artery from a needle directed caudally.

from the contrast material itself may cause spasm of the arteries. When the caliber of the catheter is too large in relation to vessel size, the frequency of spasm is increased.

Spasm may occur at the puncture site or at a distance from it and simulate arteriosclerosis, even to the slow runoff. Arterial contractions may be local over a short segment, or may be multiple and involve the entire length of the artery.[6] Wickbom and Bartley[16] noted evidence of local spasm in 39 per cent of peripheral arteriograms of patients in whom catheters were used. More often, the smaller vessels were involved and the abnormality was more frequent in females of the younger age group. In patients with Reynaud's disease, arterial spasm was present at all ages and independent of vessel size.

FLOW ARTEFACTS

Flow artefacts are produced by layering, turbulence, and retrograde injection.

LAYERING AND STREAMING

Hypertonic solutions of contrast material are heavier than blood. Slow injection in the direction of blood flow and slow circulation favor streaming and layering of contrast material in the dependent parts of horizontal vessels. Reduced turbulence facilitates layering. Contrast medium injected distant from the site of interest permits the time for turbulence to have sub-

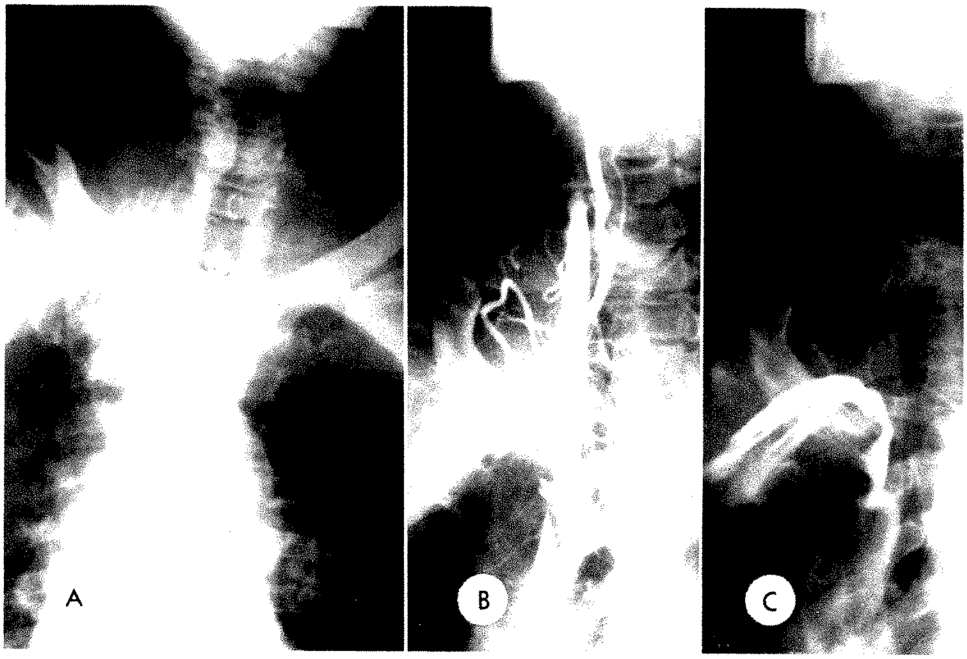

FIG. 6. (*A*, *B* and *C*) Extraluminal filling, unrecognized on a test injection, was followed by full injection, resulting in sufficient intramural deposition and injury to produce eventual occlusion of the common carotid and vertebral arteries. Intramural pooling is seen on the delayed roentgenogram. The left neck vessels were already occluded by disease and the patient died.

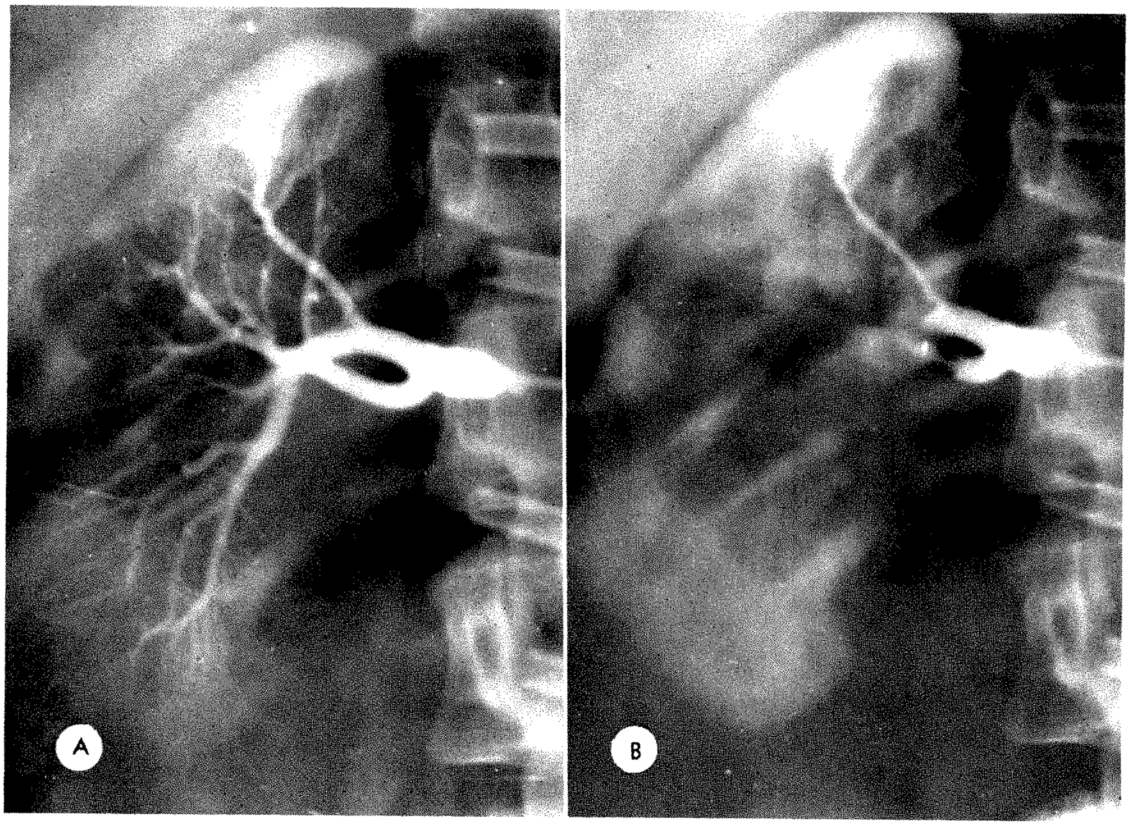

FIG. 7. (*A* and *B*) Selective catheterization of right renal artery. The sudden peripheral spasm which occurred during injection might have been mistaken for occlusion had serial roentgenograms not been obtained.

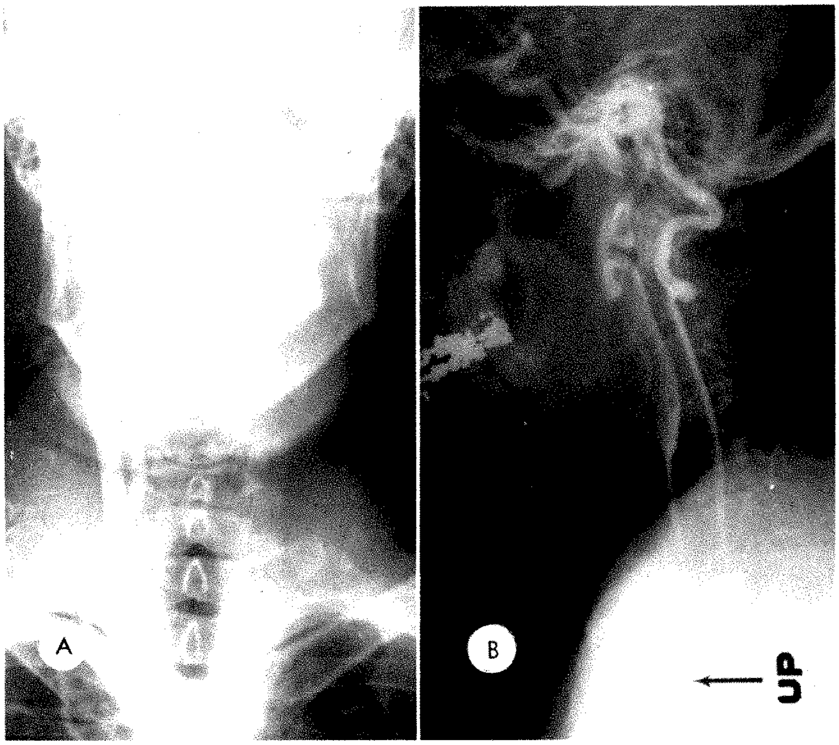

Fig. 8. Biplane supine (*A*) anteroposterior and (*B*) lateral angiograms. The horizontal beam lateral angiogram printed upright shows how layering can outline only the dependent part of a vessel. On anteroposterior roentgenograms incomplete filling of the vessel with contrast medium may not be recognized.

sided so that layering is complete when flow reaches the point of interest. It is not possible to detect the unseen anterior portion of a vessel in an anteroposterior supine projection when layering outlines only the lower half of the lumen. In the lateral view, the layering is recognizable (Fig. 8, *A* and *B*).[2]

TURBULENCE

Turbulence is implicated as the etiology for simulated aortic insufficiency when force of aortic supravalvular injection is thought to prevent complete closure of valve cusps during diastole. The force of the jet is presumed to hold open the valve at the end of systole, permitting opacification of the left ventricle (Fig. 9). Under special conditions (Fig. 10), turbulence also prevents detection of significant aortic insufficiency.[1,17]

RETROGRADE INJECTION

Retrograde injection may simulate block at the point where flow turns back the injected column of contrast material upon itself (Fig. 11, *A–D*).

POSITION ARTEFACTS

Position artefacts include compression effects, resulting in the appearance of simulated occlusion[4] or slow runoff, and gravity effects that conceal or reveal abnormality.

COMPRESSION EFFECTS

The prone position may narrow or occlude intra-abdominal grafts. Slow flow of blood through an intra-abdominal aneurysm may be seen in the supine position. The prone position may produce further slowing of blood flow through an aneurysm simulating reduced runoff because the increased abdominal pressure in this position may cause narrowing of the aneurysmal lumen.

The skeletal position sometimes causes compression of vessels, such as subclavian artery occlusion that occurs with a raised arm.

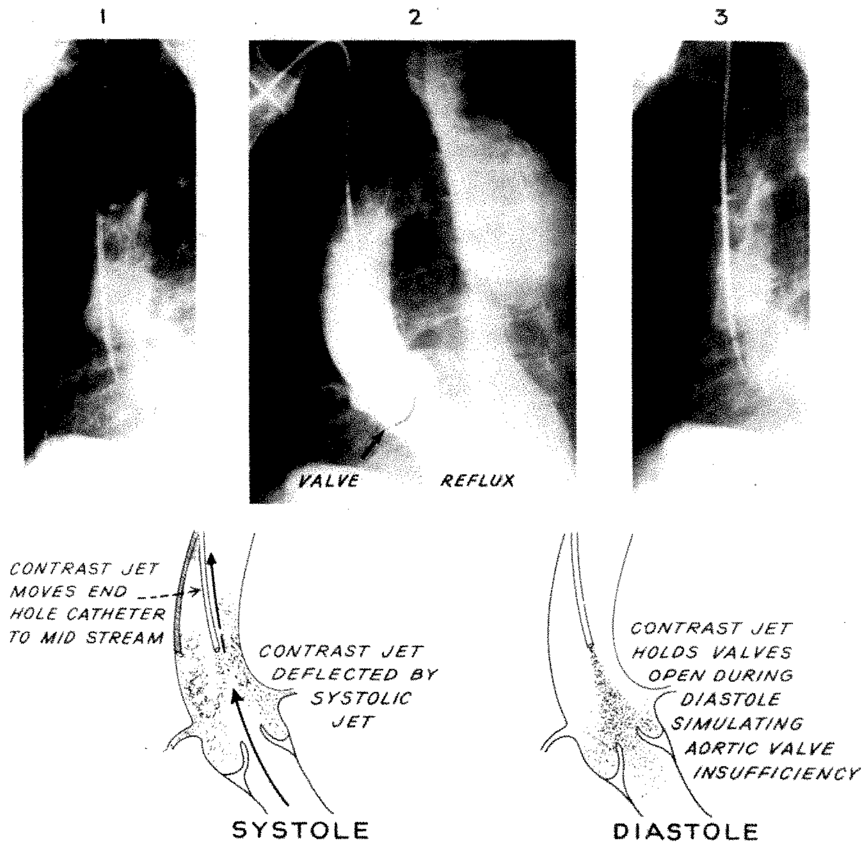
1
2
3
VALVE
REFLUX
CONTRAST JET MOVES END HOLE CATHETER TO MID STREAM
CONTRAST JET DEFLECTED BY SYSTOLIC JET
SYSTOLE
CONTRAST JET HOLDS VALVES OPEN DURING DIASTOLE SIMULATING AORTIC VALVE INSUFFICIENCY
DIASTOLE

FIG. 9. Selected roentgenograms obtained with a rapid changer are retouched to show the position of the aortic valve (clearly evident on the original roentgenograms). Although the tip of the end-hole catheter is seen above the level of the valve, a marked reflux was demonstrated. Apparently, the jet of injection held the valve slightly open at the end of systole. The patient, who had an aortic arch aneurysm, died later and a postmortem examination proved that the valve was normal.

GRAVITY

In supine position, the effects of gravity can result in obscured abdominal vascular disease. When mobile viscera are dependent and supported by the horizontal posterior abdominal wall, their vessels may coil and overlap so that vascular outlines are not well seen. Stretching of the vessels in the upright position, or, more conveniently with inspiration, reveals abnormalities formerly obscured. When unseen arteriosclerosis or fibromuscular hyperplasia is suspected, stretching of the renal vessels is particularly useful in uncovering hidden abnormalities.[9] The effects of gravity in venograms are well known and have been described elsewhere.[3]

TECHNIQUE ARTEFACTS

Artefactual shadows of processing and technique are the same as those occurring with other roentgenographic studies and are beyond the scope of this presentation. The subtraction artefact should be stressed because the specialized technique of subtraction is particularly used in angiology.[14,18] The subtraction method of superimposing diapositive nonvascular matched films upon negative angiographic films to remove the extraneous nonvessel shadows may artefactually create or conceal abnormality. Slight changes in the superimposition may create or remove this kind of artefactual abnormality with great subtlety.

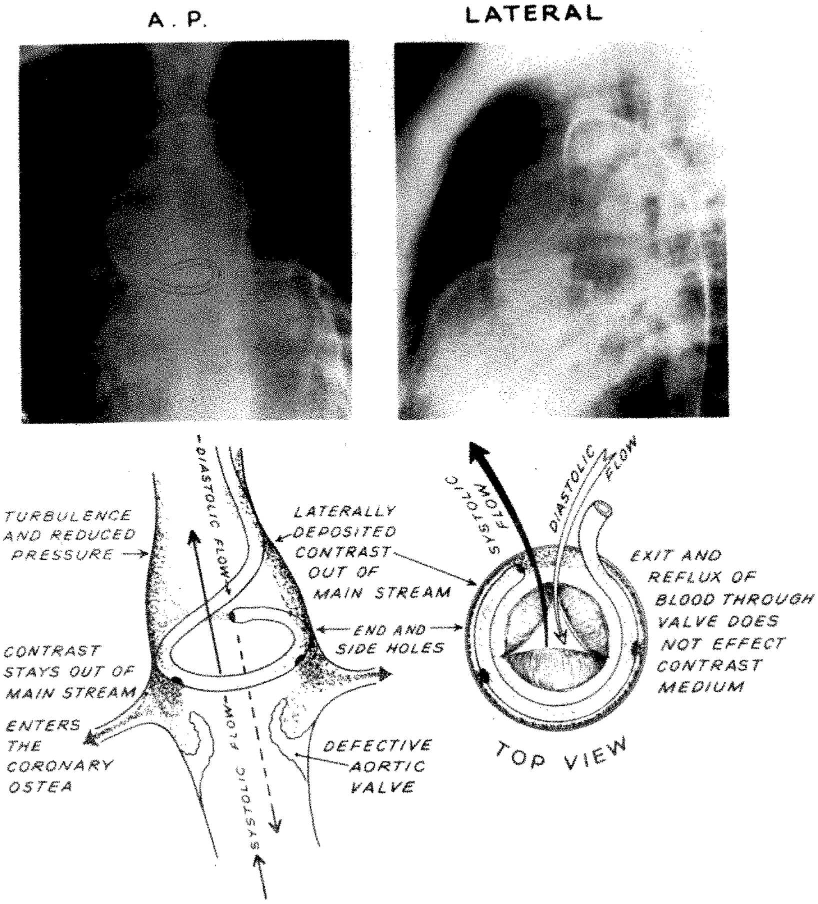
A. P.
LATERAL
DIASTOLIC FLOW
TURBULENCE AND REDUCED PRESSURE
LATERALLY DEPOSITED CONTRAST OUT OF MAIN STREAM
END AND SIDE HOLES
CONTRAST STAYS OUT OF MAIN STREAM
ENTERS THE CORONARY OSTEA
SYSTOLIC FLOW
DEFECTIVE AORTIC VALVE
SYSTOLIC FLOW
DIASTOLIC FLOW
EXIT AND REFLUX OF BLOOD THROUGH VALVE DOES NOT EFFECT CONTRAST MEDIUM
TOP VIEW

Fig. 10. Selected from biplane roentgenograms obtained with a rapid changer. The supravalvular injection was made through a loop catheter after unsuccessful attempts to pass the catheter into the left ventricle for physiologic studies. No reflux was seen. The clinically suspected but roentgenographically undetected aortic insufficiency was later proved surgically.

SUMMARY

Roentgenographic vascular artefacts of instrumentation, flow, position, and technique are classified and discussed with some examples given from experience with more than 1,200 vascular studies. The understanding of the diagnostic limitations of roentgenographic vascular techniques which in themselves create, simulate, or conceal abnormalities is an important part of vascular roentgenology.

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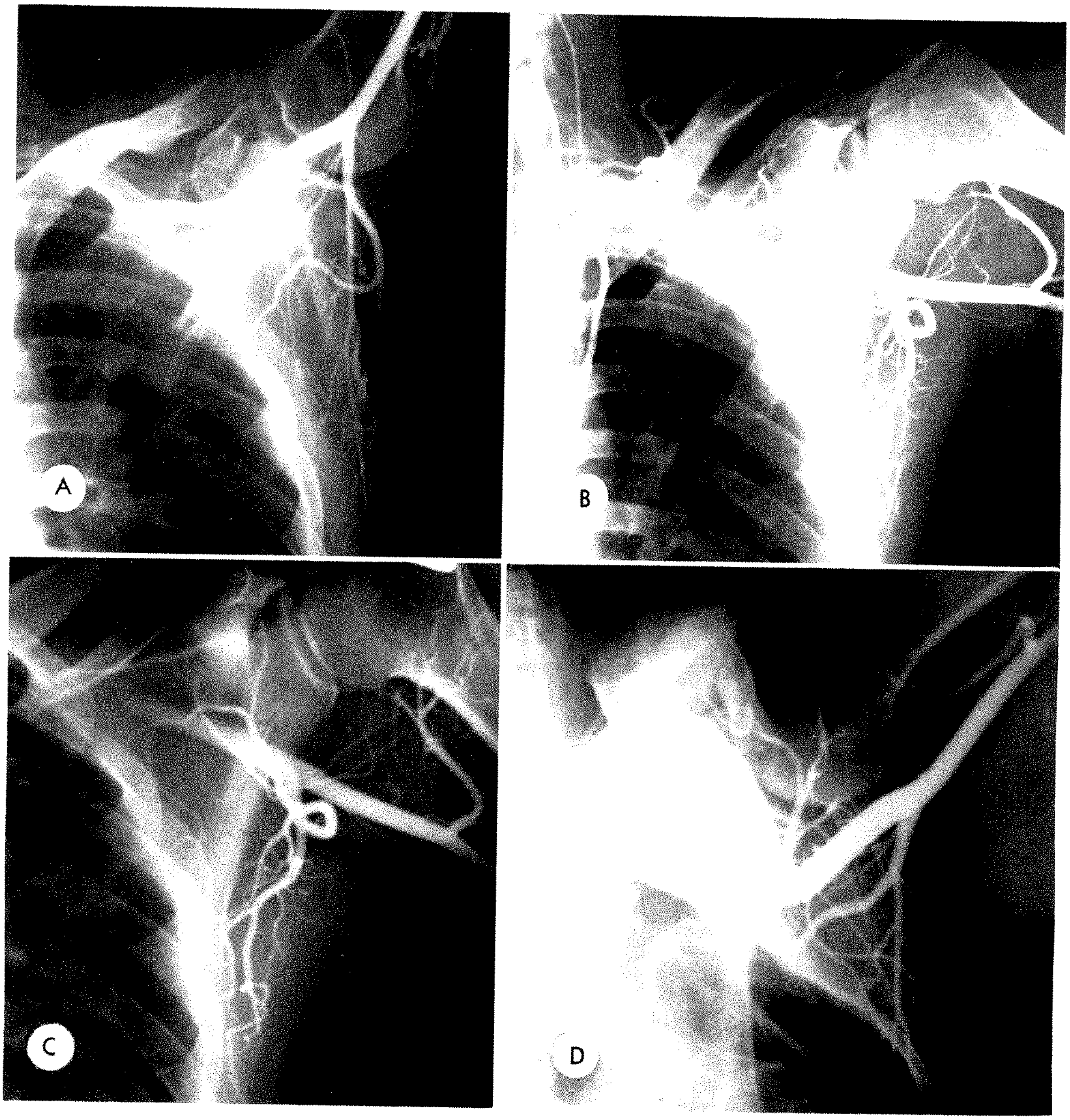

Fig. 11. (*A* and *B*) Retrograde brachial arteriograms obtained with the arm raised suggested scalenus anticus block which disappeared when the arm was lowered. (*C* and *D*) Repeat retrograde study of the brachial artery with the arm down now suggests a block in this position. This proves that the findings on the initial study were related to low retrograde injection pressure and were not the result of scalenus anticus block. The pressure varied on each hand injection and occlusion was simulated when pressure was insufficient to overcome the forward blood flow.

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PERCUTANEOUS TRANSFEMORAL ARTERIOGRAPHY*

AN ANALYSIS OF THE COMPLICATIONS IN 1,000 CONSECUTIVE CASES

By MORDECAI HALPERN, M.D.
NEW YORK, NEW YORK

PROGRESS in medicine is dependent on the analysis of scientific methods; the purpose must be understood and the results evaluated. In a short period of time the field of modern angiography has rapidly matured due to the extensive application of various catheter replacement techniques. There now exists the roentgenologic capability of selectively opacifying and visualizing all the vascular systems of the body. In the past several years, many puncture sites have been recommended for the introduction of arterial catheters into specific arterial systems. Perhaps the most generally useful approach to the various arterial systems has been the percutaneous transfemoral route. Because of the explosive development of catheter arteriography and the potential hazards involved in all intraarterial roentgenologic examinations, it would seem both useful and pertinent to present our experience in 1,000 consecutive percutaneous transfemoral retrograde catheter arteriographies.

Undesirable sequelae of the percutaneous catheter replacement technique[1,5,7,10,23,35,39,49] are connected with certain phases of the procedure. The arterial puncture site theoretically permits damage to the arterial wall, and bleeding, thrombosis, embolism, arteriovenous fistulae, aneurysm and infection have all occurred. The risk of certain of these complications is enhanced by the catheter, its characteristics, the manner with which it is manipulated and the overall time during which it remains in the vessel. The handling of the guide wire is also a factor in arterial wall damage and instances of breakage of the wire have occurred.[11,12] Damage to the parenchyma of various organs is related to contrast media. Artificial ischemia or obstruction of blood flow may be disastrous. It is imperative, therefore, that, if the tremendous diagnostic value of this method of examination is to be realized, the risk involved in this type of study be well within acceptable levels for a diagnostic examination.

MATERIAL AND METHOD

During the past 3 years 1,000 percutaneous catheter arteriographies have been performed by the Department of Radiology of The New York Hospital—Cornell Medical Center. A critical examination of this experience is the purpose of this report. In Figure 1 the salient features of the group of patients who underwent percutaneous catheter arteriography are presented. The age distribution shows a significant number of patients (52.6 per cent) in the 5th, 6th, and 7th decades of life. Thus, atherosclerosis and peripheral vascular disease were frequently encountered though their presence was not necessarily the indication for the examination. Thirty-nine per cent of the total group were studied due to the presence of clinically important hypertension. These two segments of the patient population fall into the high risk category.

The technique under discussion is percutaneous transfemoral retrograde aortography and selective catheter arteriography, also referred to as the modified Seldinger technique or catheter insertion by cannula replacement. The historical development of modern arteriography, a review of the literature and discussion of the arterio-

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* From The New York Hospital—Cornell Medical Center, New York, New York.
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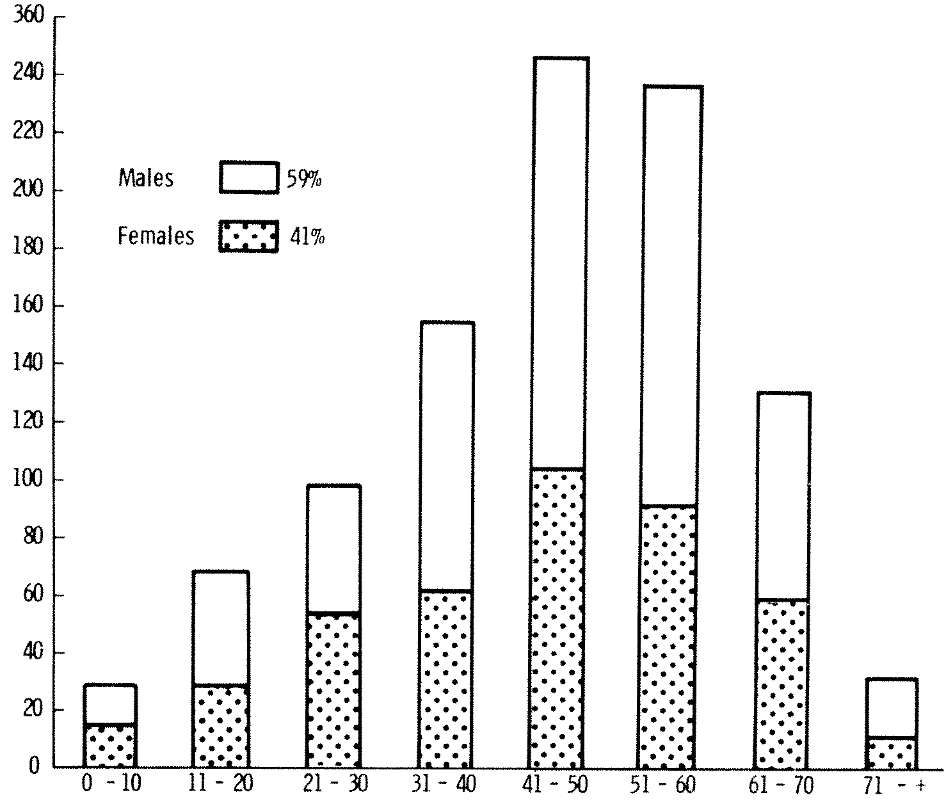

FIG. 1. Age and sex distribution of 1,000 patients in whom percutaneous transfemoral catheter arteriography was performed. The abscissa indicates the age in years and the ordinate the blood pressure in Hg. The age ranged from 4 to 84 years. There were 39 per cent hypertensive patients and 8 per cent Out Patient Department patients.

graphic puncture and other catheter techniques are amply documented in the literature and will not be discussed.[16,21,42,44,45] However, certain facets of the technique, the personnel involved and its mode of performance in a large academic medical institution all have significant bearing on the understanding of the results of this experience.

The percutaneous method of arterial catheterization has been performed throughout this period of time without modification of the basic step by step procedure as outlined in the literature.[25,48] The femoral artery is unquestionably the preferable site for percutaneous puncture. The brachial and axillary vessels are secondary sites. Only transfemoral studies were reviewed. Double catheter arteriography has been performed through both femoral arteries in order to allow catheterization and simultaneous visualization of the celiac and superior mesenteric arteries for complete visceral arteriography.

Since complications of the technique are related, at least in theory, to the amount of catheter manipulation required, Figure 2 and Table 1 present a condensation of this information. The records do not reveal the number of instances requiring catheter exchanges in the performance of selective arteriography. While a single preformed catheter may be suitable for more than one vessel, there are times when it does not adequately conform to the vascular anatomy of the patient and must, therefore, be replaced in order to achieve one's goal. This is also true of the group of multiple studies which includes, for instance, bilateral selective renal arteriography, abdominal aortog-

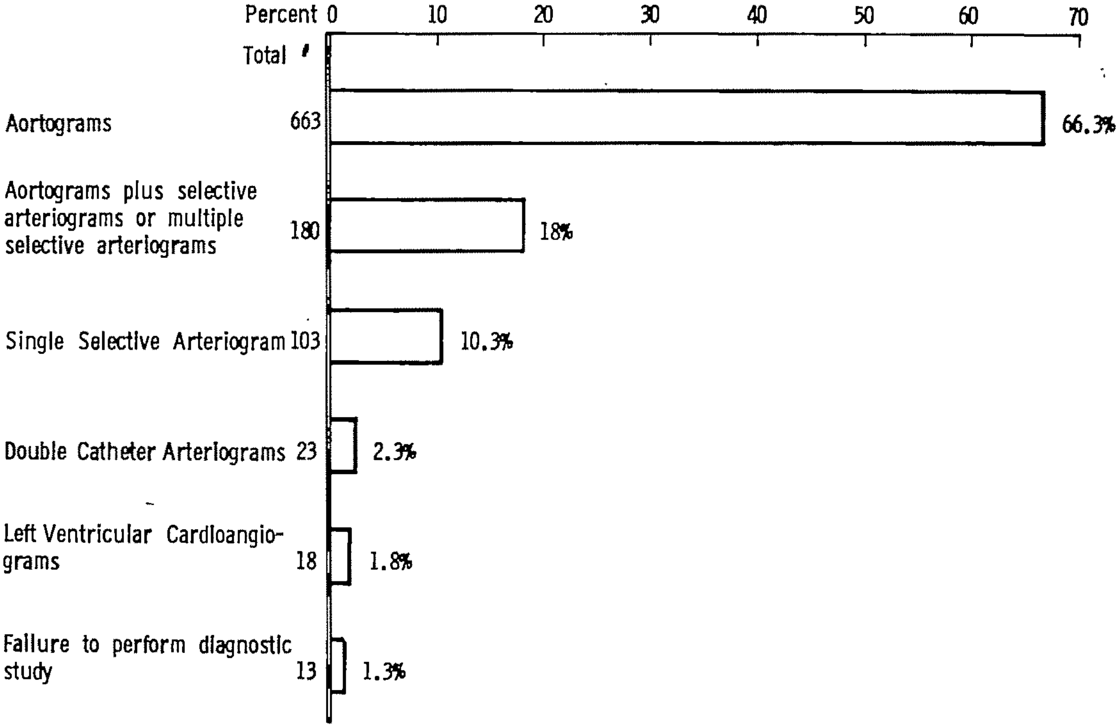

FIG. 2. Type of examination in 1,000 patients in whom percutaneous transfemoral arteriograms were obtained.

raphy in conjunction with selective renal arteriography or thoracic aortography in addition to selective examination of one or both carotid vessels. Several complex studies included consecutive catheterization of the orifices of more than two arteries. Catheter manipulation has been felt to be a prime factor in increasing the incidence of certain of the complications.[40]

The great majority of these roentgeno-

TABLE I

SELECTIVE ARTERIOGRAPHIES

Total Attempts		407
Failures	5.1%*	
Single Renal Arteries		228
Failures: (1) main renal artery	5	
(2) supplemental artery	4	
Celiac, Superior and Inferior Mesenteric Arteries		78
Failures: (1) celiac axis	3	
(2) superior mesenteric artery	2	
(3) double catheter study (only 1 vessel catheterized)	2	
Carotid and Subclavian Arteries		62
Failures: (1) carotid artery	4	
Vertebral Arteries (left only)		18
Failures:	1	

* Obstructed or occluded orifices not tabulated as failures.

graphic studies has been performed by a single investigator. While there is no question that this is a safe procedure and simple to perform and since many potential complications must be avoided, it has been our policy to delegate to one individual the primary responsibility for these studies. This has permitted a rapid buildup of specialized knowledge, skill and experience. Subsequently, selected individuals with a particular interest in cardiovascular radiology have been trained in these techniques. While a complete understanding of the arteriographic procedures and their interpretation is necessary for each trainee, there has been no pretense that each would be trained as an expert in catheter arteriography. Residents have been allowed to participate as assistants to the senior members of the cardiovascular team and those with sufficient maturity, judgement, interest and manual dexterity have been permitted to gain actual experience under the guidance of the senior radiologist. This stringent protocol has been dictated by an awareness that a thorough knowledge of technique is necessary if high quality diagnostic studies are to be obtained with minimal risk. It is our opinion that the information derived from such studies is directly proportional to the quality of the roentgenographic examination. Specialized diagnostic studies in radiology require the assumption of full patient responsibility by the radiologist. Inexperience, carelessness and lack of full appreciation of what this simple technique entails can result in many unnecessary complications.

It would seem sufficient but necessary to generalize in regard to equipment employed. The arterial needles and guide wires employed have been manufactured by Kifa though we employ the thin-walled B-D pediatric arterial needle for performing percutaneous catheterizations in young children. In the main, the catheter materials employed have been the Ödman-Ledin opaque catheters (Kifa). The features of these catheter materials for arteriography have consistently proved their excellence in our hands in comparison with others. The choice of the individual item is of course dictated by the type of study. Catheter size and the texture of the surface of the catheter material have also been mentioned as factors in the complications of the technique. The red Ödman catheter (O.D. 2.0 mm., I.D. 1.15 mm.) has been used for the majority of renal aortographies and selective renal arteriographies; the yellow Ödman catheter (O.D. 2.85 mm., I.D. 1.5 mm.) for visceral and thoracic arteriography; and the gray Ödman catheter has been an alternate in thoracic and coronary arteriography, arteriography of the great vessels of the arch and in selective left ventriculocardioangiographies.

The technical aspects of percutaneous catheter arteriography pertinent to the discussion demand some comment. The sterilization of equipment follows usual surgical principles. The nature of the catheter material requires sterilization in bactericidal solutions and, therefore, thorough washing in physiologic saline before their introduction into the arterial tree. The optimum character of the operating field has not really been established. There are inherent limitations in the sterile fields that can be established outside of a distinct surgical operating room area. When this is coupled with the simplicity of percutaneous cannula insertions, a wide technique variation results. A degree of cleansing and draping of the puncture site area is probably universal. However, the study is performed by some with cleansed but bare hands,[46] others with sterile surgical rubber gloves extending to the opposite extreme of total masking and gowning of all participating personnel. Local infection and septicemia is almost unheard of and is probably responsible for some inadvised laxity in good technique. It is difficult to categorically state what the details should be but one must always realize that a foreign body is being inserted into the blood stream. Our policy has been to follow the established principles of surgical asepsis.

Clot formation within or at the tip of a catheter, embolism and thrombosis are a triad of potential occurrences that have resulted in the use of heparinized saline solution as an intermittent or continuous flush. Many investigators adhere to this principle but some have abolished the use of heparin because of bleeding problems. No proof really exists that heparin prevents these complications. The questionable value of the intermittent or continuous drip is best exposed by watching the peculiarities of flow from a catheter that has both end and side holes. On the other hand, the metallic guide wire will provide a focal point for clot formation and, therefore, it should not be maintained in an intraarterial position for an excessive period of time.

There are additional background factors to be mentioned before proceeding to a detailed discussion of complications which have occurred. Dexterity in the manipulation of the arterial needle, care in handling the guide wire, finesse in the insertion of the catheter and skill in its guidance are basic principles which must be successfully mastered. The handling of the arterial vessels and the use of intraarterial foreign bodies demand the greatest caution. The care of the puncture site following removal of the catheter cannot be overstressed. Accurate digital pressure in this area prevents bleeding and enhances closure of the arterial puncture wound. Improper pressure may permit extensive bleeding on one hand or, in theory, promote thrombosis on the other. Experience and complete understanding of the care required by a punctured artery which has served as an entrance point for a catheter that has been in place for a variable period of time are absolutely essential for the proper performance of the technique.

Finally, the postoperative patient care is important. The procedure is in fact, an operative one, no matter how minor it may be. Generally, all patients need local pressure to the puncture site for periods of time that may vary from 8 to 15 minutes or more. Most of the time, the patient is placed in the supine position from 1 to 4 hours with a pressure dressing over the puncture site at the termination of the study.

Complications due to contrast medium must be mentioned though detailed discussion of this complex problem is beyond the scope of this paper. Broad experience with cardiovascular contrast media affords a feeling of confidence and security in the performance of arteriographic studies.[41] The proper concentration and quantity of the opaque material employed for the various catheter studies are sufficiently described in the literature not to require repetition here.

DISCUSSION OF COMPLICATIONS

Since reports in the literature do not reflect any unanimity of opinions as to just what represents a complication of a diagnostic procedure, some definition of the term must be supplied as the basis for further discussion. A fortuitous circumstance detrimental to the patient's status which arises as a result of the method is certainly a complication. This may be minor and temporary or of a major magnitude.

Furthermore, one must distinguish reports presented in the literature under the broad heading of complications of aortography from those which pertain to the specific technique under discussion.[2,6,9,13,14,31,36,37] Most of the former bear reference to translumbar aortography and intraarterial studies performed through arteriotomies. In this connection the greater simplicity of performance, the greater safety and the greater versatility of the catheter replacement technique should be contrasted with the other modes of examination.[1,10,13]

BLEEDING COMPLICATIONS

Hematoma and bleeding require first consideration. Our postprocedural format obligates one of the individuals of the diagnostic team to examine the patient within a few hours following the completion of the study. A return is made to the patient at the end of approximately 24 hours. The area of puncture is examined and the status of the extremity and arterial pulses is assessed. The findings are noted in the patient's record, whether normal or otherwise. This supplements routine orders to nursing service and house staff which call for inspection of the vascular status of the extremity each half hour for a 2 hour period after the study. Of course, individual interpretation enters into the picture, but to establish some degree of constancy, the author alone has reviewed all of the patients' charts. It became readily apparent that no useful purpose could be served by categorizing mild local ecchymosis and/or mild induration as a complication. Discrete extravasations of a volume estimated to be below 20 cc. of fluid were likewise discounted unless they were in intimate relation to the artery. This lesson resulted from the one experience with a false aneurysm. This will be discussed subsequently. Currently, all local hematomas are diffused away from the vessels following removal of the catheter. This requires a simple rotary motion during application of digital pressure. While the end result is one of somewhat more diffuse ecchymosis, it effectively destroys the foundation on which a false aneurysm may form. A summary of vascular complications is given in Table II. Some of these have occurred coincident with removal of the catheter and hence are the result of insufficient digital pressure. Bleeding at this time is usually recognized quickly, and the institution of corrective measures almost always prevents a possible complication. One large hematoma was treated with local hyaluronidase while another received only local conservative treatment. The rapidity of dissolution of hematomas has varied and in no instances were there any permanent complications. These individuals all showed persistence of good pulses. In one patient fever and transient incomplete venous obstruction were noted; however, gradual subsidence without residua occurred. Ödman[40] has made mention of postangiographic fever and of femoral vein thrombosis secondary to large hematoma formation. Undetected bleeding may result in shock.[15] The phenomenon of delayed bleeding is a distressing finding and the reason for it is not clear. It has occurred in patients between 40 and 60 years of age. Admittedly, in some there have been arteriosclerotic changes out of proportion to chronologic age. In one individual, the pressure dressing became displaced and an extensive hematoma developed. Though the pulses remained good, apprehension as to the possibility of a retroperitoneal hematoma resulted in surgical exploration at the end of 48 hours, at which time no bleeding point was identified in the femoral artery. One 48 year old individual underwent double catheter visceral arteriography without incidence only to spurt blood from the left femoral puncture site when she sat up at the end of 36 hours. A pressure dressing was reapplied and the groin and leg pulses were essentially normal 12 hours later. Among the group studied on an outpatient basis, there was one unconfirmed report of a 21 year old hypertensive woman who bled copiously while performing household duties on the day following the examination. An additional patient in the same clinic category is currently being observed for a slowly resolving hematoma which was minimal at the termination of the study but increased considerably 1 day later.

Bleeding must be discussed relative to other phases of the procedure. Once an artery has been punctured a potential bleeding site has been established and proper attention must be paid to it. Improper puncture may militate against passage of the guide wire or of the catheter. Repeat punctures of the same vessel should only be done with an awareness of the increased complication risk. Objection to deliberate through and through penetration of the vessel by the arterial needle is on the same basis. Not only is it unnecessary for proper needle placement, but it adds additional trauma to the artery. Furthermore, while insertion of the needle into the femoral artery close to the inguinal ligament may be done inadvertently, puncture of the posterior wall above this location distinctly enhances the chance of a retroperitoneal hematoma.

Following removal of the cannula, with the guide wire in place, insufficient digital pressure will permit free bleeding. While the blood generally flows along the tract formed by the needle and wire and does not usually spread into the tissues locally, such bleeding may be excessive and dangerous and should be prevented. Anticoagulant therapy and the existence of abnormal bleeding traits are considered to be contraindications to the procedure. However, the study has been performed in a significant number of patients in the first group without complication. Certainly, one is justified in assuming an increased risk when the situation requires it just as surgery is performed under the same conditions.

On rare occasions, oozing can be seen around a catheter that has been placed in position. This is most often due to arteriosclerotic disease of the vessel. Ready control of such minor blood loss is accomplished by the gentle application of pressure. There have been times when this was required during the total time the catheter was in place so that the examination could be concluded. Similar ooze may occur during manipulation of catheters for selective arteriography. Once again, this does not necessitate abandonment of the study and need not result in hematoma formation. A high percentage of hematomas has been listed as a concomitant of selective study as has the use of the larger type catheter (O.D. 2.85 mm.). While in theory this seems logical, the statistics of this series do not confirm the point. There appear to be unpredicatable factors peculiar to some vessels that allow bleeding episodes. In one middle aged patient, a small catheter (O.D. 1.17 mm.) was readily inserted atraumatically for renal aortography and yet considerable blood flow occurred around it. The operator chose to discontinue the procedure. There were no resultant complications.

In reviewing the literature, it became readily obvious that numerous reports as to

Table II

ANALYSIS OF COMPLICATIONS

	No. of Cases	Mild and Transient	Severe and Transient	Permanent
A. Bleeding or Vascular Complications				
I. Hematoma (significant)	12	10	2	0
(1) delayed bleeding without hematoma	2	2	0	0
(2) delayed bleeding with hematoma	2	2	0	0
(3) uncontrolled bleeding with catheter in place	1	1	0	0
II. Thrombosis	4	0	0	4
(1) loss of pulse*	1	0	0	0
III. Embolism	0	0	0	0
IV. False Aneurysm†	1	0	1	0
B. Complications Related to Guide Wires or Catheters				
I. Fractured Guide Wire Tips‡	2			
II. Intramural Aortic Passage of Catheter	2			
C. Miscellaneous				
I. Renal Damage	0			
II. Infection	0			

* Operation at 48 hours did not show thrombus.
† Operation performed.
‡ Removed from femoral sheath.

bleeding complications exist. Adeney and Fraser[3] report no complications in 103 patients; Aagaard *et al.*[1] report 29 hematomas in 535 studies; and Treleaven and Copestake[49] list 75 hematomas not requiring treatment in 76 catheterizations. Dotter[15] has reported 4 hematomas causing discomfort in a group of 110 patients and, in addition, 1 patient previously mentioned had undetected bleeding which resulted in shock. Nonetheless, this investigator is one of the advocates of performance of the study on an out-patient basis and, in fact, states that early ambulation is a preventative against bleeding. Observation of these individuals is made until they are able to walk freely without bleeding. A pressure dressing is utilized as a deterrent to bleeding. The greatest experience in this technique comes from abroad where temporary pressure and a period of rest represent the total after care of many patients. Similarly, the same approach is used by most cardiopulmonary laboratories in this country, employing the procedure for left heart catheterization. However, an unknown percentage of these patients shows significant local induration with blood extravasation. The patient's local discomfort is considered to be an acceptable part of the procedure. Since most of this group receives prophylactic antibiotics following cardiac catheterization, no estimation of postangiographic fever can be made. The crux of the matter lies in the answer to the question—what degree of bleeding and local tissue reaction is to be considered a complication of arterial catheterization? Summation of the problem is not difficult. Table II shows that significant bleeding is rare when the examination is carefully conducted. In addition, transient small hematomas are also most often preventable. One should strive for the greatest perfection possible and not accept a minor complication as an inherent characteristic of the procedure.

Thrombosis and embolism have been reported with this form of percutaneous arteriography.[23,35,39,49] The risk of such occurrence is presumably related to the risk of damage to the arterial wall. Theoretically, selective arteriography should increase this possibility. Vascular spasm would also play a role. This complication will most often involve the site of introduction of the catheter and is, therefore, related to the technical finesse of the operator. Lapses in technique are all avoidable. Multiple erratic punctures and forceful introduction of the guide wire or catheter will definitely cause undue trauma to the vessel wall. Luke and McGraw's[35] experience has caused them to stress the role of too vigorous compression of the femoral artery and to express the opinion that it is wiser to accept moderate bleeding rather than employ pressure over the puncture site at the termination of the examination. There is no question that excessive pressure may be damaging and that proper closure of the arterial wound can be accomplished with modest, accurately placed digital pressure. The hypertensive and arteriosclerotic patients are definitely a higher risk and may require pressure from 12 to 20 minutes at the termination of the study.

The 4 patients in this category of complications represent an incidence of 0.4 per cent of thrombosis; no embolic phenomena have occurred. An additional patient was studied 5 years following aortic valvulotomy at which time bilateral femoral arteriotomies were performed. A bounding pulse was present just below the inguinal ligament. More distally, the pulses were markedly diminished to absent though the leg was asymptomatic. Following the percutaneous arterial catheterization, there was loss of the original bounding common femoral pulse with some coolness of the leg, which changes persisted for 48 hours. At this time exploration revealed heavy arteriosclerotic deposits without evidence of fresh thrombus. The procedure did, of course, cause some local vascular alteration in an already compromised vessel. A return to this pre-examination state might well have resulted from conservative treatment.

In this connection there are several points of considerable interest and importance. To begin, a considerable number of patients examined by this procedure has been studied with reference to peripheral vascular disease and in this group many are also hypertensive. For instance, Aagaard and co-workers' series[1] contained 269 patients with peripheral vascular disease of the total group of 535 patients. Figure 1 shows the number of hypertensive patients in our series and the age groupings of the patients studied. Most of the patients subjected to selective renal, selective carotid and visceral arteriography fall into the older age group. While hemorrhage, embolism and problems with the retrograde passage of the catheter would seem to be predominant in this group, experience suggests that thrombosis may be primarily a problem in the cardiac group of patients. While operative proof is not available, there has been permanent loss of femoral pulse in one patient who is a 35 year old male with supravalvular aortic stenosis while the other three complications were noted in patients with rheumatic heart disease and severe mitral insufficiency, 2 of whom did not survive operation for mitral valve replacement. Three of four complications presented by Mandelbaum and Shumacker[39] were in patients who underwent combined cardiac catheterization and angiography with resultant arterial thrombosis. One of Luke and McGraw's[35] patients also had rheumatic heart disease. In recent years, ischemia and thrombosis developed in 2 children with cardiac disease who underwent open femoral arteriotomy at The New York Hospital. In 39 of 125 cases Vengsarkar and Swan[60] reported some definite effect on the peripheral arterial circulation though no permanent tissue damage resulted. Jones *et al.*[29] have discussed major complications following the insertion of femoral input catheters at the time of open heart surgery. It is interesting to note that in this group there were no complications in the patients with congenital heart disease while 2 deaths resulted from arterial complications in the group of 35 patients with acquired heart disease. The evidence suggests that there is some peculiarity of the vessels in individuals with acquired heart disease. The occurrence of thromboembolic phenomena in this group of patients is well recognized.

In theory, selective arterial catheterizations are thought to increase ischemic arterial complications. Once again, though exact figures are not available, the cardiac patient regardless of age appears to represent the greater hazard.

It is further felt that the absence of even a single episode of embolism in this series attests to the fact that, while apprehension regarding embolism may be present, it need not be confirmed in practice. Undetermined factors may always exist, but strict adherence to certain principles will continue to be the prime means of preventing the complications of percutaneous catheter arteriography.

Additional vascular complications comprise diminished arterial circulation and/or delayed return of pulses. Such transient phenomena require careful patient observation, but no definite treatment as a rule. Severe vascular spasm has also rarely occurred. If it becomes intractable during the procedure, papaverine therapy can be employed with success. Such spasm following catheter removal must be differentiated from thrombosis before instituting treatment since the former is most often self limited.

INFECTION

Previous mention of postarteriographic fever has been made with the presumption that this was secondary to hematoma formation. The potential for local infection and for septicemia also exists. No such episodes occurred in the experience being reported. In some series, protocol calls for routine postprocedural bactericidal treatment. We have restricted its use to the high risk patient, primarily cardiac patients with valvular disease. Similar prophylaxis has been utilized in diabetics and in patients whose recent medical history reveals a specific bacterial infection. Cutaneous staphylococcal infections are felt to represent a contraindication to the examination. Likewise, study of cardiac patients recently treated for septicemia or bacterial endocarditis is deferred until such patients are considered acceptable for cardiac surgery.

FALSE ANEURYSMS

False aneurysms have been reported as important sequelae of arteriography.[23,35] The personal case to be added to the literature merits some detailed attention. The experience gained and lessons learned have been invaluable. The patient was a severe hypertensive male of 40 years of age who was the 94th case in the series of 1,000 and represened the 10th out-patient examination. The study was easily performed and completed within a minimum period of time. There was no bleeding at the puncture site. Routine care consisted of a pressure dressing weighted with a sandbag and maintenance of a supine position for 1½ hours. The patient was discharged from observation with instructions to avoid stress for 12–24 hours. These warnings were ignored. Mild exercise took place within 8 hours and vigorous exercise (skiing) was indulged on the following day. At the end of 48 hours the patient was seen, at which time examination revealed diffuse, superficial ecchymosis over the right thigh and right lower quadrant of the abdomen. In addition, a hematoma estimated at 30 cc. was present over the femoral puncture area. The ecchymosis soon disappeared. During the ensuing 3 months the patient continued periodic skiing and his physician reported no change in the extent or character of the hematoma. Shortly thereafter, there was sudden enlargement of the groin mass with obvious pulsation. At operation, the femoral artery showed a clean, V-shaped laceration from the previous arterial catheterization which was closed by a single suture. There were complications of this operative procedure due to a wound infection but these are not pertinent to the present topic. This episode caused considerable consternation as to the advisability of continuing studies on an out-patient basis, especially in the high risk group of patients. This has, however, been continued cautiously since the problems and economics of hospitalization are difficult. One of the patients with a delayed hematoma is in this group. On the constructive side, immediate steps were taken in an attempt to ensure that no patient would be subjected to strenuous activity within 24 hours of study. There has been one additional episode of delayed bleeding in an hypertensive out-patient. Gentle massage of all hematomas has been instituted so that they are diffused from the region of the femoral triangle. It is believed that the persistence of a mass over the puncture site for a prolonged interval is best handled by exploration, evacuation of the hematoma and repair of the artery, if required.

The report of an arteriovenous fistula between the saphenous vein and the femoral artery by Pierce and Ramey[43] is of interest. The course of the arterial needle would normally avoid the venous structures. Occasionally, there is inadvertent puncture of the femoral vein during arterial catheterization. The same set of circumstances may result where the vein itself is the intended puncture site and the artery is accidentally perforated. Probably, this combination of punctures in association with extravasation of blood provides the basis for an arteriovenous fistula. Pierce and Ramey's experience appears to be unique but the hazard is real. In the present series, deliberate cannulization of both the femoral artery and vein has been performed. The indications have been first, the necessity for assessment of the integrity of the inferior vena cava or the renal veins as in the case of renal trauma and, second, to obtain samples of both arterial and renal blood for chemical analysis. Perhaps understanding of the increased risk has permitted such elaborate investigations to be performed without difficulty. It should also be noted that aftercare of a venous catheter study calls for patient ambulation to prevent the possibility of venous thrombosis in contradistinction to the pressure dressing and rest given to patients who have undergone arterial catheter studies. In Gregg's[23] report, the femoral arteriovenous fistula or aneurysm at the puncture site occurred under most unusual circumstances. Wound infection occurred around a catheter that had been left *in situ* over a period of 5 days, during which time chemotherapeutic perfusion for chondrosarcoma was in progress. Only then did the arterial complications become manifest.

CATHETER AND GUIDE WIRE COMPLICATIONS

Complications related directly to the guide wire insertion and to the retrograde passage of the guide wire and catheter are of great importance. Boijsen and Feinstein[10] mention the possibility of breakage of the tip of the guide wire and of perforation of the aortic wall by the catheter, but no such accident was recorded in their 1,000 cases. Cope[12] and others[1,5,11,23,47,49] have experienced fracture of the guide wire tip both in the perivascular tissues and in an intravascular position. This has led Cope[12] to suggest the sealing of the wires with teflon and caused Aagaard *et al.*[1] to revert to the use of a Cournand No. 4 cardiac catheter with a movable metal stylet in place of the flexible guide wire. However, the use of a catheter as a guide wire has apparently resulted in the severance of such a catheter during its withdrawal through the cannula, suggesting that the guide element will remain as a potential hazard. Two such acccidents are recorded in Table III and in each instance retrospective analysis brought some definite lapse in technique to light. Both episodes were quite early in total experience at which time 120 cm. PE 160 guide wires were employed for renal aortography though the catheters were but 45 cm. in length. Such wires are more susceptible to wear and tear than the shorter length (80 cm.) wire or the PE 205 wire. There is little doubt that, in addition to improper technique, the wires themselves were defective through prolonged use. The exact life of this piece of equipment cannot be categorically stated. It must be carefully inspected and discarded if there is any suspicion as to its integrity. An improper positioning of the tip of the arterial cannula may result in an obstructed passage of wire, but an intact wire can be withdrawn without fracture. In each instance in these 2 patients, the tip of the wire became fixed in the tissues of the femoral sheath. While re-

Table III

ANALYSIS OF PROBLEMS OF TECHNIQUE

		No. of Cases	Per Cent of Total
I. Failure to Puncture Femoral Artery		2	0.2
II. Obstructed Passage of Catheter		21	2.1
A. Contralateral Puncture Performed	13		
B. Contralateral Arteriotomy Performed	2		
C. Bilateral Failure	2		
D. Unilateral Failure (contralateral attempt not made)	4		
III. Examinations Discontinued		2	0.2
A. Bleeding around Catheter (Table II)	1		
B. Onset of Angina and Brachycardia Prior to Injection	1		
IV. Failure to Catheterize Desired Vessel (See Table I)		21	5.1

moval was not absolutely necessary, the foreign body was retrieved under local anesthesia so that a more complete understanding of the technique could be obtained. The posterior location of the tip furthered the belief that through and through perforation of the artery was not a good procedure. It also confirmed the opinion that the angle of incidence of the arterial needle in relation to the artery should be as acute an angle as possible. This is in opposition to the technique of perpendicular puncture of the artery with subsequent depression of the cannula to free its tip within the lumen of the artery. Low cost expendable guide wires have proven to be totally unsatisfactory in our experience. Equipment must be of the highest quality and cost a secondary consideration. The correct passage of a guide wire is essentially without sensation. Any degree of resistance or sense of friction requires immediate evaluation of the cause. There are no substitutes for experience, good technique and good judgement. Handling of the catheter during its retrograde passage requires the same degree of finesse. In this connection, it might be well to mention that the tactile sense of the status of the catheter tip may be lost when the subcutaneous tissue about the puncture site does not permit frictionless passage of the catheter. A small stab wound has been adopted for passage of all yellow and gray Ödman catheters and in all Negroes regardless of the size of the catheter employed.

Dissecting aneurysm of the aorta[47] and dissection of the aortic wall[24] have been reported as complications of catheter aortography as has perforation of the iliac artery following catheter insertion through a femoral arteriotomy. The true incidence of these severe and potentially fatal complications is not clear. Lang's[33] recent survey of 11,402 percutaneous retrograde arteriographies uncovered 7 fatalities, representing a 0.06 per cent mortality rate.

In 1 patient of the group being reported, passage of the catheter was forced in the region of the aortic bifurcation. When final positioning at the L-1 level was achieved, there was free flow of blood. Subsequent injection of contrast material with a pressure injector resulted in an intramural extravasation which caused severe but transient back pain. There were no further sequelae. Many of the patients investigated have had significant atherosclerotic disease with or without abdominal aneurysms and/or hypertension and many have had peripheral vascular disease. The percentage has not been as high as that in other reported series.[1,14] With the use of image intensification and with broadening experience, most of the attempted studies have been completed. Difficult retrograde passage of the catheter has been performed in several patients without compromising the safety factor. No arteriosclerotic plaques have been lifted free to become emboli and no clinically significant vascular trauma of any type has been noted. During the process of manipulation of a catheter and/or guide wire past protruding arteriosclerotic plaques, test injections of 1 cc. of contrast material have revealed minor trauma to the arterial wall on a few occasions. Such situations demand immediate abandonment of the study. In one such case, the catheter was withdrawn from the aorta into the iliac artery and successfully repositioned. The remainder of the examination was uneventful. Nonetheless, such action is not recommended. There is, however, no contraindication to a second attempt through the opposite femoral artery after an interval of a few days. It is well to re-evaluate such patients before a repeat examination and to determine whether a different point of entry, such as the axillary artery, is indicated, or whether another method of aortography is in order. The vascular surgeons at The New York Hospital frequently request intravenous abdominal aortography as the preferred method of study for patients with peripheral vascular disease and aorticoiliac atherosclerosis since the exquisite detail provided by catheter aortography is not required for full preoperative evaluation of the patient. Translumbar aortography has been completely discontinued at this center in favor of the percutaneous retrograde catheter method.

False passage of the guide wire or catheter is reported in the experience of several authors. Others have encountered similar difficulties which have led them to seek other means of arteriography. Yet in skilled hands, large series of cases have been accumulated within which considerable numbers of patients were studied for peripheral vascular disease without any problem arising. Figure 2 and Tables I, II and III confirm the fact that proper examination can be performed in the greatest number of patients without complication. In only 13 of 1,000 cases were the diagnostic studies not completed.

During the initial 50 examinations performed, the records reveal complete failure to cannulize the femoral artery in 2 patients: Patient 3 was an adult and Patient 33 was an obese 14 year old girl. Subsequently, patients as young as 4 years of age and obese patients up to 380 pounds have been examined without undue difficulty. Guide wires can be inadvertently directed into the deep circumflex iliac artery. After insertion of a few centimeters of wire, an abrupt halt is noted in association with sharp pain. This is quite characteristic and recognizable. Withdrawal of the guide wire into the cannula followed by a 180 degree rotation of the cannula or threading of the cannula further into the femoral artery is the most commonly successful maneuver to overcome the situation. During the initial phases of this study, 6 such instances occurred. With increased experience, this is now rarely a problem though the examination may have to be performed through the contralateral femoral artery. In 2 patients, one 8 and one 11 years of age, the percutaneous procedure was abandoned for this reason and contralateral arteriotomy was performed. This again was early in the series. There have been 9 patients in whom passage of the catheter was obstructed through the right femoral artery, which is used preferentially, while the examination was readily performed through the opposite femoral vessel.

Obstruction to the passage of the catheter has occurred in atherosclerotic iliac arteries with or without aneurysms or in the lower abdominal aorta in 9 patients. In 1 individual an unsuccessful attempt was made from the opposite side. These patients all underwent intravenous aortography. Complete preoperative evaluation of thoracic aneurysms has, at times, required both retrograde transfemoral and antegrade right axillary arteriographies.

There have, of course, been times when selective catheterization of an arterial orifice was the prime purpose of the examination and failure resulted. These studies were completed by either thoracic or abdominal aortographies utilizing the original catheter or following replacement with a catheter of more appropriate shape. This most commonly involves the great vessels of the arch of the aorta. While 7 such instances are listed, this is not the entire picture. On one hand, the presence of a disease process obstructing the vessel orifice can completely negate selective study. Such cases are not listed with the other experiences. On the other hand, there have been occasions when the patient's symptomatology was so vague that it was difficult to determine just which vessel should be selectively catheterized. At such times, brief attempts at selective arteriography were made and if not immediately successful thoracic or abdominal aortography was employed rather than further selective attempts which would require a change of catheter.

Rounding out the problems of guide wire and catheter passage, there has been one instance in which a guide wire was passed through one common iliac artery into the contralateral vessel due to advanced disease at the aortic bifurcation. A catheter was also noted to have taken the same course. Under image intensification, the course was readily corrected. With the assistance of this equipment, failures in catheter passage are rather uncommon. Previous to the use of image intensification, there was 1 adult patient in whom the catheter could not be made to traverse the aortic arch into the ascending aorta. Finally, 3 of 18 attempts to cross the aortic valves for selective left ventriculocardiography resulted in failure. In this connection 2 deaths must be listed, realizing that they are not specifically complications of the percutaneous transfemoral arterial method. Both patients were Class IV cardiacs due to rheumatic heart disease. The first patient succumbed to intractable ventricular fibrillation which ensued simultaneously with the passage of the guide wire into the left ventricle. Subsequently, only J-shaped catheters have been employed for this purpose. Following the filming procedure, a second patient registered ventricular tachycardia which changed to fibrillation. Prompt defibrillation and routine emergency procedure corrected the rhythm disturbance but the patient died 18 hours later. Fibrillation is an inherent hazard in selective left ventricular studies, and when it occurs it is difficult to pinpoint the exact cause.

CONTRAST MEDIUM

Initially, it was stated that this report did not properly encompass the subject of contrast medium. It is not the intention, however, to detract from the extreme importance of this subject. No one can justifiably perform any type of angiography without first becoming completely conversant with the properties of opaque arteriographic media.[4,17,18,19,20,27,28,34,51] In reviewing this literature, one must be careful not to confuse the toxic effects of contrast media now in discard, erroneously presuming that these are the properties of modern media. A case of renal atrophy following renal arteriography in which sodium acetrizoate contrast medium was utilized was reported by McCallister *et al.*[38] in 1962. Caution in the use of this medium had been previously recommended by Lance *et al.*[32] in 1959. In our series of cases diatrizoate salts have been used almost exclusively in concentrations of 50–90 per cent. Recently, in a number of cases sodium iothalamate has been employed since certain of its characteristics are an asset in arteriography. This substance permits more favorable catheter flow rates than accomplished with 90 per cent diatrizoate salt. There have been no signs of renal damage in the patients undergoing either aortography or selective renal arteriography. There has been no evidence of damage to visceral organs. Cerebral reactions in neck and intracranial studies have been transient and of the same general magnitude as found in carotid arteriography.

A comparison of the complications following retrograde transfemoral cerebral angiography with those of direct carotid angiography in patients with occlusive cerebrovascular disease reveals a neurologic complication rate of 4.5 per cent as contrasted with 11 per cent in the latter group. The group analyzed may be too small to permit valid statistical conclusions, but it does suggest that the retrograde catheter technique is safer than direct puncture in this group of high risk patients.

CONCLUSION

This personal series of the first 1,000 consecutive percutaneous transfemoral catheter arteriographies performed at The New York Hospital—Cornell Medical Center is made up of a patient population that other investigators might anticipate. Many patients (39 per cent) were studied specifically for hypertension. Many of the older adults were examined in order to demonstrate the presence of carcinoma. In a small group of patients abdominal angina was the reason for study. Dissecting aneurysms and saccular aneurysms were other indications in a small percentage of cases. Occlusive vascular disease, especially cerebrovascular disease, represented still an additional segment. The series thus includes a large group of high risk patients due to hypertension and arteriosclerosis. To this category, patients with acquired cardiac disease should be added.

While problems of technical procedure have occurred in a statistical total of 7.4 per cent of cases, successful diagnostic conclusion of the examination was obtained in all but 1.4 per cent of the entire group. Total significant complications as defined earlier were met 2.4 per cent of the time and of these 0.7 per cent required some corrective operative procedure and none appears to have done permanent damage to the patient. It should be noted that insufficient time has passed for full assessment of the thrombosed vessels and the repaired false aneurysm. Should these limbs become symptomatic in time, this will have to be attributed to the procedure.

The series includes a considerable number of selective examinations without an increased rate of complication. It should be re-emphasized that the size of the catheter employed has also not been a factor which affected the percentage of complications. No renal damage has been noted and there have been no complications due to contrast medium.

Not only does it appear that percutaneous catheter arteriography carries a lower rate of significant problems than those seen with open arteriotomy, but the patient morbidity is far less. Limited experience utilizing the catheter for intracranial studies in patients with occlusive cerebrovascular disease suggests that this is a superior method of approach. Finally, comparative studies of retrograde catheter arteriography and translumbar aortography[31] seem to reveal little significant difference in the serious but nonfatal complication rate of these diagnostic studies.

Analysis of this series points out the tremendous value of the versatility of the technique. At the same time, the inherent hazards and technical problems are less than those found in other methods of arteriography. The precise roentgenographic anatomic detail achieved is so invaluable to the care and treatment of the patient that it more than justifies the study of high risk individuals. In many instances, a substitute method is not available.

The over-all success is attributable to various factors among which the most important are: (1) a high respect for a simple procedure that entails passage of a foreign body through an intraarterial course; and (2) rigid adherence to sound technical procedure.

SUMMARY

An analysis of the complications and the procedural problems which occurred in 1,000 percutaneous transfemoral catheter arteriographies performed during the period of 1960–1963 by The Department of Radiology of The New York Hospital—Cornell Medical Center has been presented.

The percutaneous transfemoral catheter technique is the backbone of modern arteriography. It is a diagnostic roentgenologic method that can be performed with a complication rate completely acceptable for a diagnostic technique.

In this series, the incidence of hematomas and bleeding problems was 1.7 per cent while thrombosis and loss of pulse occurred in 0.5 per cent of cases. There was 1 false aneurysm in the group and 2 instances of fractured guide wire tips. There were no episodes of infection, renal damage or embolic phenomenon.

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AN ARTERIAL SIMULATOR FOR ANGIOGRAPHIC RESEARCH*

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THE detection of stenotic arteriosclerotic lesions by arteriography is a simple and widely practiced procedure. The significance of these lesions on the other hand is often difficult to evaluate in terms of flow restriction or physiologic importance. Present arteriographic techniques yield, at best, crude estimates of flow. Other clinical methods of measuring flow in single arteries have limited application in that they can be used only during surgery.

It seems desirable to extend the usefulness of arteriography in the measurement of blood flow. To study this problem, many variable parameters of hemodynamic physiology must be controlled. In order to evaluate the relative importance of each of these variables, it was planned to initiate this investigation on an experimental model of the vascular system in which single factors could be independently studied.

In this report, the design and construction of such an arterial simulator for angiographic research are described.

PARAMETERS OF INTEREST

The specifications of the vascular simulator, the injection system and other pertinent data are presented in Table I. Verified accuracies are listed. Figure references denote sources of information in subsequent illustrations. Figure 1 shows the complete apparatus.

HYDRAULIC FEATURES OF CONSTRUCTION

The relationship of the components is best described by reference to Figure 2, a schematic drawing. The reservoir may be filled with simulated blood by using a mixture of glycerol and water to produce a

TABLE I

PARAMETERS OF INTEREST

	Specifications	Accuracies	Refers to Figure 3
Arterial Features			
Flow	0–12 cc./sec.	±3.8%	
Pressure	120/80 mm. Hg	±10 mm.	Trace 1, 3
Pulse	60–160/min.	±1.0%	Trace 2, 5
Size	.188–.375 in.		
Injection Features			
Dosage	0–50 cc.	±5%	
Duration—D.C.	0–1,000 ms.		Trace 6
Duration—Actual	varies		Trace 5
Applied Pressure	0–250 p.s.i.	±10%	
Hydraulic Pressure	0–1,000 p.s.i.	±10%	Trace 5
Relation to R-wave		±10 ms.	Trace 3, 5
Multiple Injections	1–12 times		
Roentgenographic Features			
Camera Exposure Time	shutter		Trace 3
Camera Delay	0–1,000 ms.		Trace 6, 3
Simulated Angiogram			

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* Supported by the Oregon Heart Association, Good Samaritan Hospital Research Foundation, and private contributions.

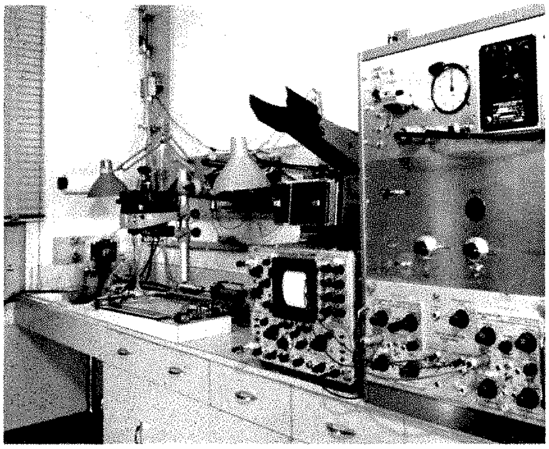

Fig. 1. Arterial simulator.

viscosity of 3.3 c. st. Two pumps move the fluid from the reservoir to the manifold (M) and then into the two artificial arteries (A and B) which empty against the pressure of standing columns of fluid.

An important feature of the system is the maintenance of a blood pressure of 120/80 mm. Hg as the flow in the test artery (A) is varied from 0 to 10 cc. per second. If a single piston pump is used, an excessively high pulse pressure results because Tygon tubing is more rigid than the human arterial system. The desired arterial pressure curve was obtained by blending the output of two pumps. P_1 is a freely rotating impeller type pump. It accounts for most of the flow (10 cc./sec.) and is adjusted by valve V_1. Pump P_1 working against the standing fluid column maintained a pressure level at 60 mm. of Hg. By using a second special two valved piston pump (P_2) with a stroke volume of approximately 2.0 cc., we achieve an output of 12 cc. per second through the two arteries.

Adjustments of the artery valves V_2 and V_3 shunt the flow from one to the other and maintain the desired variation in flow in artery A. Flow measurements are based on the filling of a volumetric burette in a measured time.

The experimental arterial segment (A) is removable. Its size and shape can be varied. The other artery (B) has an important secondary function. We refer to this as a shunting characteristic. This phenomenon is evident whenever an injection is made at a rate exceeding the acceptance rate of the artery. Under this condition the contrast agent flows in both directions.

The angiographic needles or catheters enter the artificial artery via the manifold (M). The presently used contrast agent is Prussian Blue. A Polaroid camera is used in place of the roentgenographic apparatus. The modified Shipps injector is connected by tubing producing the same resistance found in our clinical systems.

ELECTRONIC FEATURES

Signals from the hydraulic components (Fig. 2) include: R wave of the electrocardiogram (pulsatile pump P_2), arterial pressure (T_1), hydraulic injection pressure (T_2), as well as camera exposure duration.

These data are photographed simultaneously with a second camera on a Tektronix 565 oscilloscope. The slower sweep rates permit study of the events preceding, during, and following the injections. The faster sweep rates are used to evaluate small details in single injections. The built-in delay system of the upper beam of the oscilloscope is used to trigger, at any point on the pressure curve or at any time between R waves, an external circuit that starts the injection. This external circuit determines the injection duration, number of injections, and the delay from the beginning of the last injection to the roentgen-ray exposure (C_1).

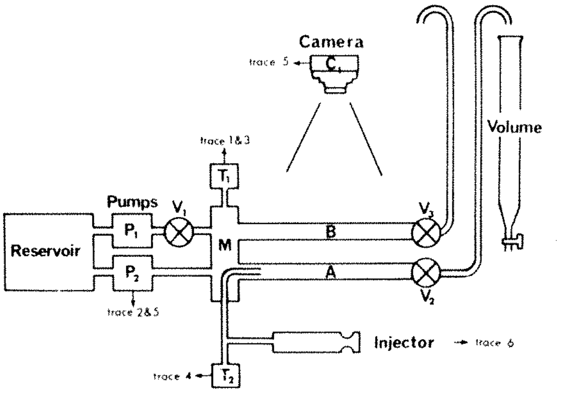

Fig. 2. Schematic drawing.

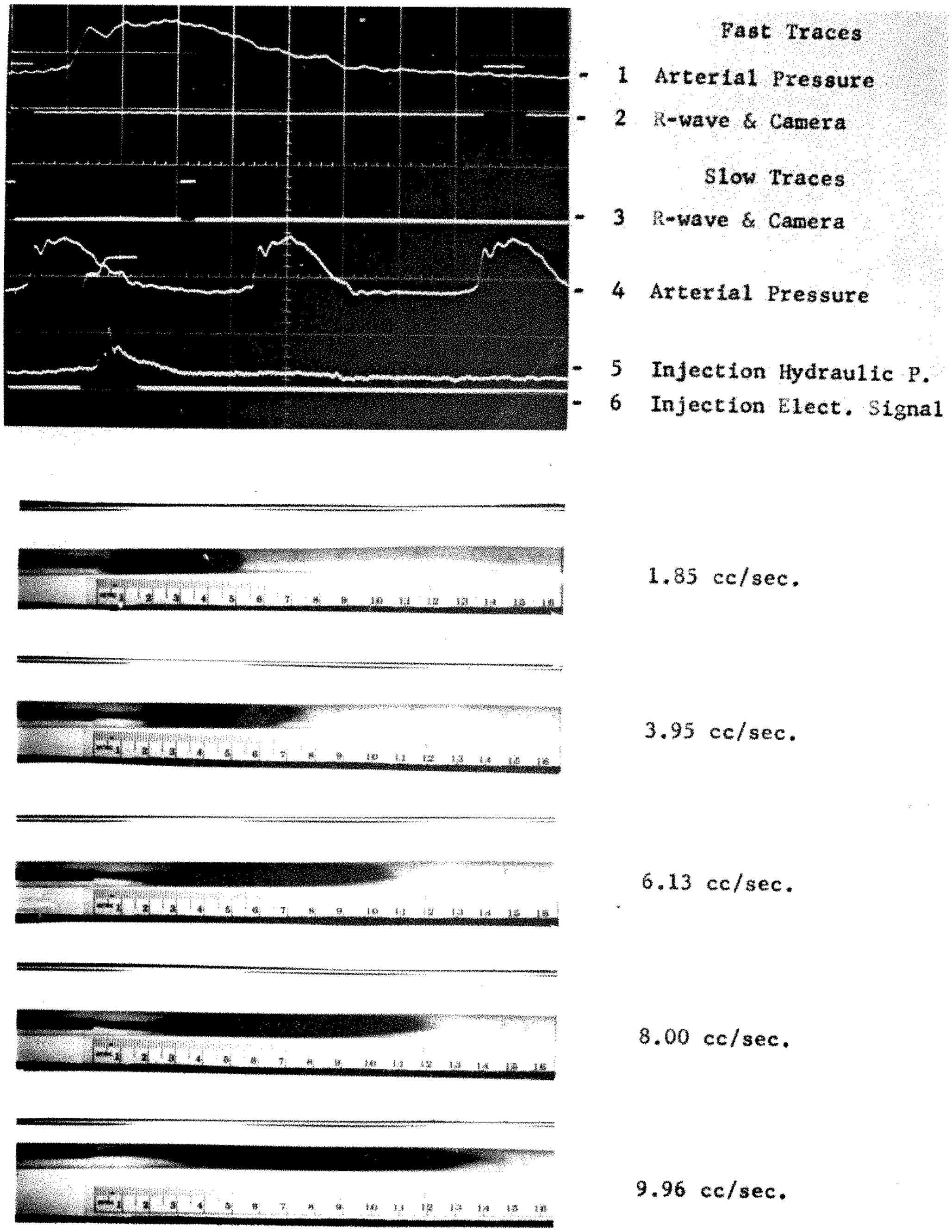

Fig. 3. Sample experimental data.

Built into the above is a system that allows calibration of the flow rates over set periods of time, or pre-set number of pulses. This allows the investigator to calibrate on each injection.

SAMPLE DATA

The apparatus produces two Polaroid photographs during each test condition. In Figure 3 the trace pattern remained constant so that only one oscilloscope trace is illustrated. The artery photographs reflect the single variable in the test, changes in fluid flow.

SUMMARY

A brief description of the major features of an arterial simulator now used in angiographic research has been presented. The ultimate objective is the design and test of methods to measure flood flow in single arteries during arteriography.

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PROGRAMMED ANGIOGRAPHY*

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SERIALOGRAMS have been a great aid to diagnostic angiography. One of the shortcomings, however, is the fixed time intervals between exposures. Our experience with the Sanchez-Perez device operating at maximum speed (0.5 sec. interval setting) resulted in a fixed program of films at 0.8–0.8–0.6–0.6–0.6 second intervals in a six film sequence. This fixed program does not adequately meet the needs of cerebral angiography. The fastest setting is desirable during the early phase of cerebral circulation and a slower sequencing later in the series is more efficient.

CLINICAL DATA IN CEREBRAL ANGIOGRAPHY

We reviewed a series of cerebral arteriograms plotting the development of optimal arterial and venous phases on the basis of corrected times. After plotting 60 consecutive injections, the pattern in Figure 1 became apparent. There was a high percentage of pathologic cases to produce variations.

Curve A in Figure 1 represents the distribution of peak arterial phases in relation to time in these 60 consecutive injections. It is apparent that ideal arterial opacification is of short duration and that therefore the first exposure should be precisely placed. Attention to the physical factors of injection is important. Using the Shipps injector at 75 p.s.i. gauge pressure, we perfuse the carotid arteries with 10 cc. of hypaque 50 per cent in 1.0 and 1.2 seconds. This perfusion time may vary depending upon changes in resistance of the connecting system. We monitor the perfusion time on each injection by using the injection time clock.

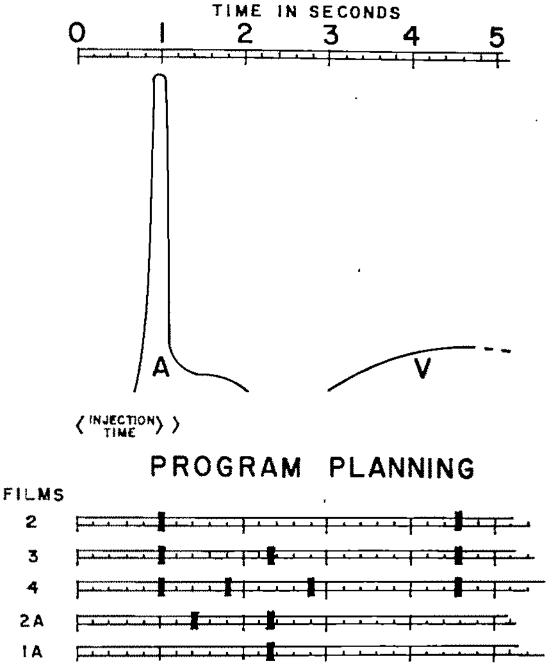

Fig. 1. Program Planning.

Referring again to curve A, between 1.0 and 2.0 seconds, it is apparent that about 1 out of 6 cases shows prolonged development of full arterial filling. Unfortunately, these cases are not readily predictable but are patients in older age groups with cerebral arteriosclerosis and/or stenoses, increased intracranial pressure, or vascular spasm associated with brain tumors or recent hemorrhages of any cause.

Curve V of Figure 1 represents the time variations of development of the venous phases. This phase may occur as early as 3 seconds but in most cases is in the 4.0 to 4.5 second range. The patients with marked retardation of cerebral arterial flow usually do not develop diagnostically useful venous phases.

From the foregoing observations, we became interested in the precise and individual selection of the time of exposure of each film in the serialogram. This led to the

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* Presented before the Pacific Northwest Radiological Society, May 9–10, 1964, Hilton Hotel, Portland, Oregon.

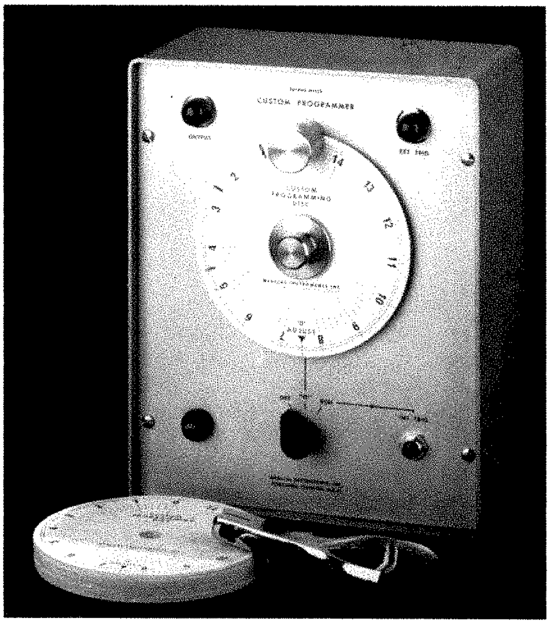

Fig. 2. Photograph of the custom programmer.

development of a custom-automatic programmer.

PROGRAMMER

The custom programmer (Fig. 2) was designed with the concept of program development in mind. It was assumed that the most desired programs for particular procedures would require considerable experience before final development. Various types of programs are likely to be needed. Once each of these is established, the programmer becomes an automatic device. The program disks act as memory cards like those used in computers. Where a variety of programs is used interchangeably, the card system will save time and obviate unknown errors of adjustment as opposed to those programmers that require individual adjustment of many switches.

The programmer operates on a photoelectric principle. A program is first laid out by marking the times of the desired output signals on the cards. A special rectangular punch then perforates the cards with 1/16 inch slots. This size hole produces a relay closure or output signal of 0.1 second. The accuracy is +.000 and −.020 seconds by oscilloscopic evaluation. Placement of slots ±.020 seconds accuracy has been achieved. Slight changes in timing are made by tracing an existing program onto a new disk, marking the changes and repunching.

The maximum rate of output signals is 6 during a period of 1 second. Variations in duration of the output signal are obtained by increasing the perforation length. The cycle length is 14 seconds, and then the programmer stops automatically until manually recycled. There are electric and mechanical lockout provisions to avoid recycling if the external triggering device (injector) is inadvertently in a triggered position.

PROGRAM PLANNING

Programming may tend to reduce excessive filming, but the number of roentgenograms needed to arrive at a diagnosis will remain a personal opinion. Here we wish to present some theories of application of programming in cerebral angiography.

Referring to programs under Figure 1, the basic cerebral angiographic program may evolve as follows: The 2-film series represents only the placement of optimal arterial and venous phases. The 3-film series shows the addition of an intermediate exposure designed to pick up a vascular stain. The 4-film series places more emphasis on the detection of an obscure arterial tumor stain. We believe that this type of program will become the basic pattern. It is acknowledged that after examining the roentgenograms of such a series other information may be desired. For example, should a subtle stain be suggested on the second film of the 4-film series, an additional program (2A) can be punched and applied in about 30 seconds.

Where the Sanchez-Perez device will not operate at closer intervals than 0.8 seconds, this additional 2-film series (2A) will result in films at .4 sec. intervals as a composite of the two series. Here the programmer increases the Sanchez-Perez performance to twice its capacity. Another programmer disk can be punched with a single hole to restudy a lesion in several different positions (1A) without overutilization of a series of films for each position. Since the programmer operates the serialograph for just the number of exposures punched on each disk and automatically stops, time is saved in not having to reload the serialograph for successive short series.

SUMMARY

Programmed serial angiography offers precise control of the time of exposure of each film in the series. The use of programming will reduce the excessive exposures of fixed serialography and increase our diagnostic accuracy in the use of the serialograph.

A custom-automatic programmer has been described.

Theories of application in cerebral angiography have been presented.

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SUCCESSFUL STERILIZATION OF A PRIMARY CARDIAC SARCOMA BY SUPERVOLTAGE RADIATION THERAPY*

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IN A PREVIOUS article the first successful use of a cardiopulmonary bypass for the removal of a primary cardiac fibromyxosarcoma was described and the literature was reviewed.[1] The technique and results of postoperative radiation therapy administered for the treatment of the residual tumor in this patient are reported here. Although death from a solitary cerebral metastasis occurred 9 months following radiation therapy, the autopsy revealed no evidence of the primary neoplasm.

REPORT OF A CASE

A 55 year old woman was admitted to the Palo Alto-Stanford Hospital on February 7, 1961, complaining of progressive exertional dyspnea associated with weakness of 3 months' duration.

The blood pressure was 110/75 mm. Hg, pulse 90/minute, and the respiration 20/minute. There was a prominent "a" wave in the jugular veins. Cardiac dullness extended 3 cm. to the left of the midclavicular line. The pulmonic second sound was softer than the aortic and was not split. A Grade 4/6 systolic murmur was heard along the sternal border and was transmitted upward to the clavicle and to the right and left neck. Liver dullness extended 2 finger breadths below the right costal margin, but the liver was not tender and no pulsations were felt. There was no cyanosis or edema. Neurologic examination was normal.

Routine blood tests and urinalysis were normal. Electrocardiograms were interpreted as showing right axis deviation with abnormal T waves in leads V_1 through V_4. Chest roentgenograms showed an increase in cardiac size when compared with those made 1 year previously. Cardiac catheterization and angiography were performed, and demonstrated a mass involving the right ventricular outflow tract, the main pulmonary artery, and the proximal branches of the right and the left pulmonary artery.

At operation, a glistening hemorrhagic lobulated tumor was found on the anterior wall of the right ventricle, involving the pulmonary artery to its bifurcation. Radical excision was attempted but complete removal could not be accomplished. The ventriculotomy was closed with Ivalon* bolster because of the friable nature of the myocardium.[1] A diagnosis of fibromyosarcoma was made (Fig. 1*A*). Postoperative radiation therapy was given with the Stanford medical linear accelerator from April 6 through May 16, 1961. Directly opposing anteroposterior fields were used to deliver the first 2,500 rads tumor dose because of the patient's inability to cooperate fully. The field arrangement was later modified to opposing right and left lateral fields with a supplemental direct anterior field in an attempt to spare the spinal cord and the pulmonary parenchyma from unnecessary irradiation. Figure 2 shows the summation isodose distribution of the four fields utilized, through which a tumor dose of 6,300 rads minimum to 7,400 rads maximum was delivered in 41 days. This course of treatment was tolerated without difficulty and no significant change from the preradiation therapy physical examination was noted. Because of the volume of lung tissue necessarily included within the treatment fields, prednisone, 15 mg./day, was given during the time of therapy and for several weeks after completion in an attempt to minimize pleuropulmonary fibrosis.

The patient remained free of cardiopulmonary problems until the time of death in January 1962, following a short course of progressive mental deterioration, hemiplegia, and coma.

At postmortem examination, no free fluid or

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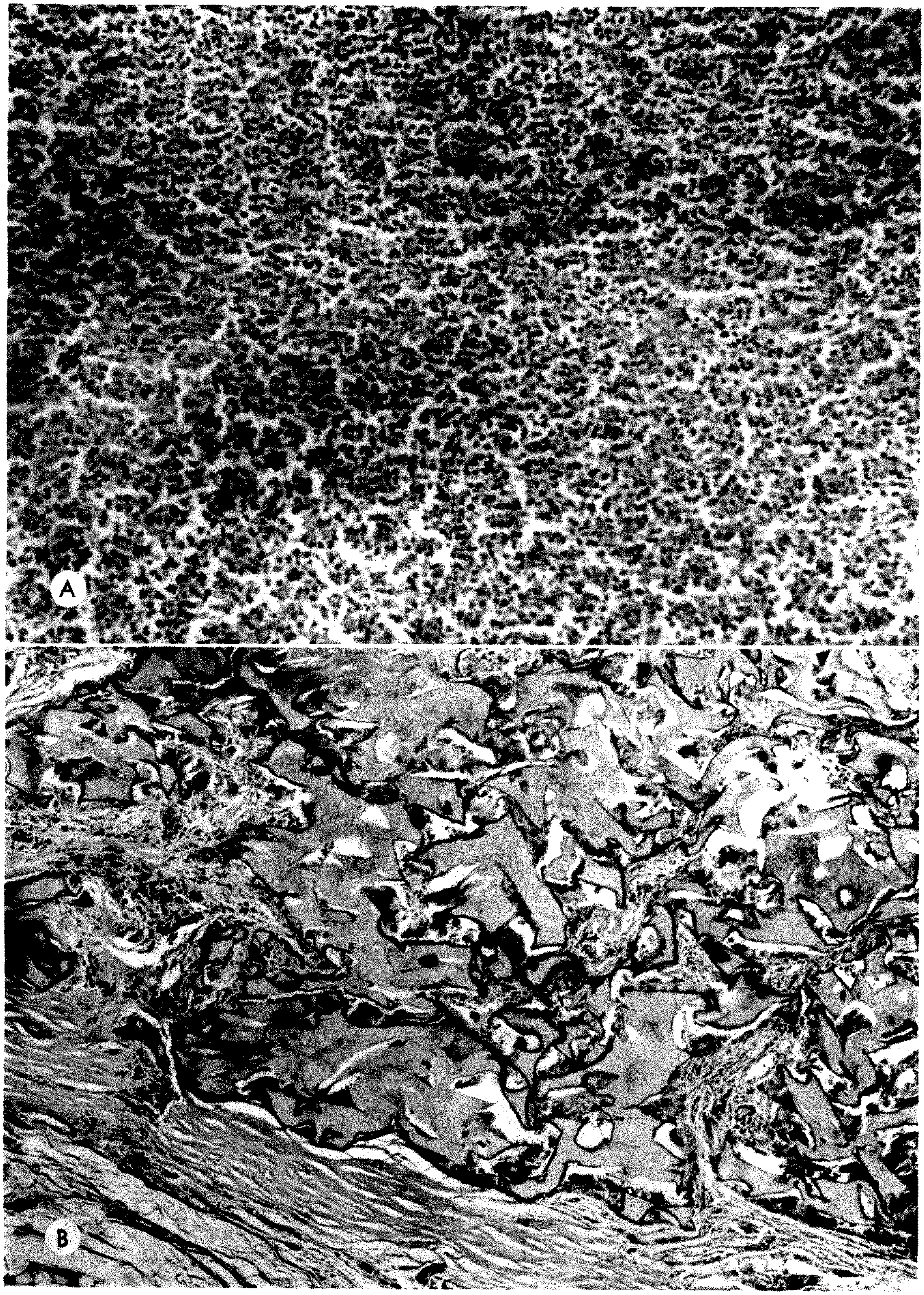

Fig. 1. (*A*) Fibromyxosarcoma involving myocardium (×54). (*B*) Ivalon bolster in myocardium without residual neoplasm (×40).

significant adhesions were noted in the pleural cavities. The pericardial cavity was totally obliterated by fibrous adhesions which were easily separated by blunt dissection. The heart weighed approximately 300 gm. The surgical site was identified easily. Sutures and the Ivalon bolster material were found where they had been placed at the time of closure of the right ventricle. The chambers were of approximately normal size, except for slight enlargement and muscular hypertrophy in the right atrium. The myocardium was of normal texture and the endocardium was smooth and glistening. Minimal atherosclerosis, without evidence of occlusion or constriction, was found in the coronary arteries. Fibrosis of peri-aortic tissues was present.

Multiple areas of thickening of subpleural tissues were noted in the right lung, the largest being a 12 cm.×3 cm. anteromedial band. A few similar changes were found over the left lung, but the parenchymal tissue was otherwise normal.

The brain weighed 1,530 gm. and was moderately swollen with flattened sulci and narrowed gyri. A 3 cm.×2.5 cm.×3 cm. diameter tumor was found within the thalamus indenting the left half of the floor of the third ventricle. No other nodules were discovered.

The microscopic examination of the heart showed the cardiac fibers to be normal. No residual tumor could be identified (Fig. 1*B*). Adventitial fibrosis was present in the aorta and there was severe fibrosis with hyalinization in the pericardium. Study of the lungs revealed pleural, perivascular, and peribronchial fibrosis with partial obliteration of the alveolar spaces.

A single, well-circumscribed but not encapsulated tumor composed of large multinucleated cells was found in the left hemisphere of the brain. The nuclei were irregular and hyperchromatic with frequent nucleoli. The appearance was similar to that of the tissue obtained from the primary tumor at the time of cardiac surgery, and not characteristic of a primary brain tumor (Fig. 3).

DISCUSSION

Radiation therapy has not been utilized in most cases of primary heart tumors, probably because of the "radioresistant" state attributed to the normal heart and

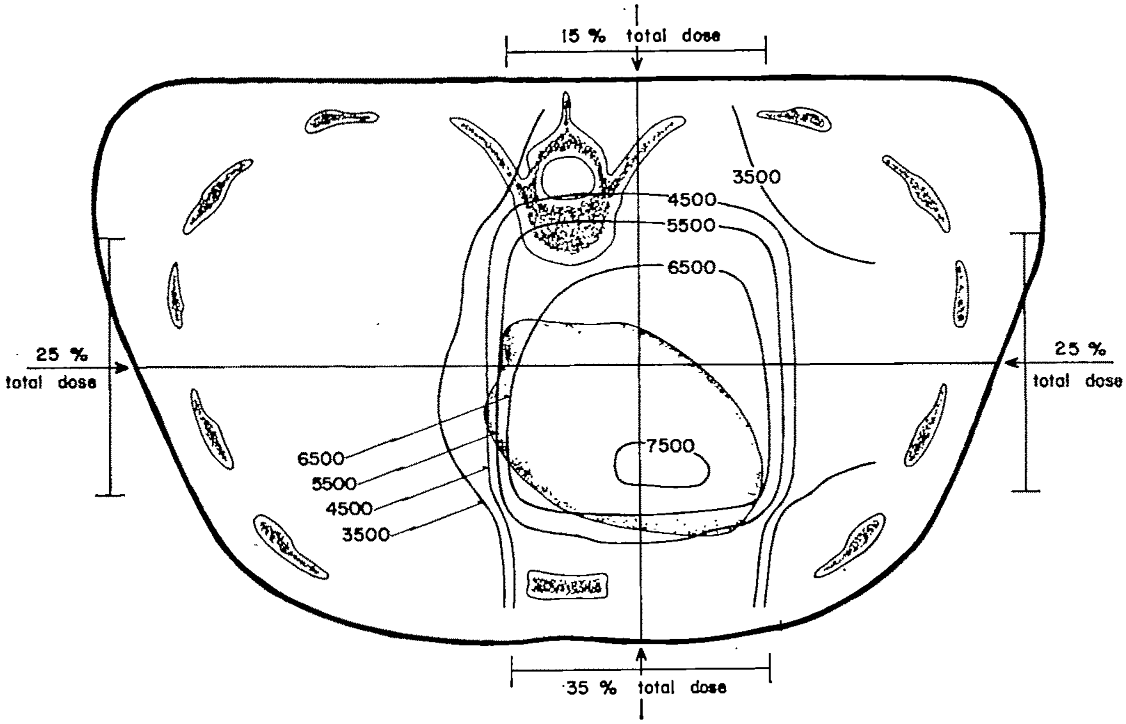

Fig. 2. Summation isodose distribution of the 4 fields (4.8 mev.).

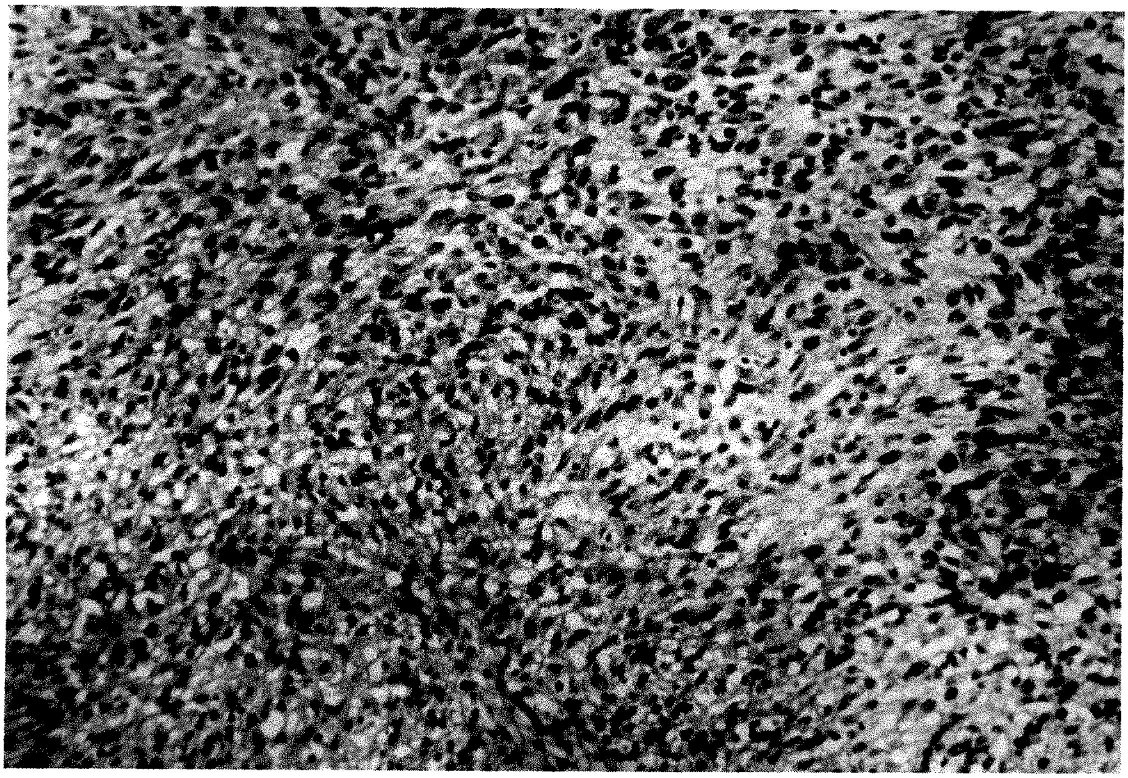

Fig. 3. Metastatic fibromyxosarcoma to thalamus (×71).

assumed to exist for the tumor. However, should the tumor prove more radiosensitive than the normal heart, this would constitute an excellent reason for employing radiation therapy. Haythorn *et al.*[2] presented a patient thought to have Hodgkin's disease with pulmonary parenchymal metastases. Three of the nodules were treated to approximately 2,000 r without response; no other details of therapy were given. Postmortem examination revealed primary fibromyxosarcoma of the heart, originating in the first portion of the pulmonary artery and extending along the pulmonary tree, with multiple pulmonary metastases.

Congestive heart failure with venous obstruction is the usual mechanism of death in patients with cardiac tumors. When metastases occur, they are primarily to the lung, other sites being relatively uncommon.[5] It is apparent that the tumor remaining after cardiac surgery was sterilized by radiation therapy in the present case. In contrast to other reports[3] of myocardial damage when the heart has been included in the radiation field of other tumors, there was no evidence of damage to the cardiac muscle 7 months after completion of irradiation.

Radiation therapy should be considered for those patients in whom resection of a primary cardiac sarcoma is not feasible or successful.

Prednisone was used in addition to careful treatment planning in an effort to diminish pulmonary fibrosis, which might have occurred after high dose radiation therapy. No clinical pulmonary symptoms developed, and chest roentgenograms 4 months after radiation therapy (Fig. 4, *A* and *B*) disclosed no evidence of pulmonary fibrosis. Some microscopic evidence of fibrosis was present at postmortem examination, and this was most marked in the regions which received the highest dose.

The possible response to radiation therapy of the solitary metastatic lesion in the thalamus is a subject for speculation.

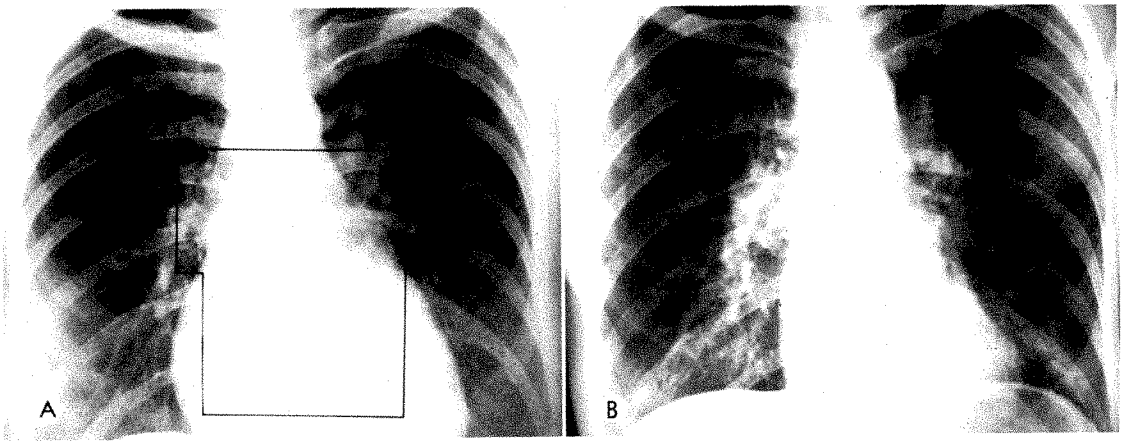

Fig. 4. (*A*) *February, 1961:* Preradiation therapy chest teleroentgenogram. Note increase in cardiac diameter and prominence of right ventricular outflow tract. (Anterior radiation therapy field indicated.) (*B*) *June, 1961:* Four months later. Note decrease in cardiac diameter and lack of pulmonary fibrosis.

SUMMARY

1. Residual cardiac fibromyxosarcoma was sterilized by high dose radiation therapy without evidence of functional cardiac damage.

2. Death was due to a solitary metastasis to the thalamus.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Forty-seventh Annual Meeting: Roosevelt Hotel, New Orleans, La., April 8–10, 1965.

EDITORIAL

THE EVOLUTION OF CEREBRAL ARTERIOGRAPHY

CEREBRAL arteriography, first described 33 years ago, is now an established diagnostic procedure. In recent years many different methods and techniques have been devised. A short review of some of the more widely used of these may, therefore, appear advisable, particularly since there is at present a tendency to reject or deny advances made in cerebral arteriography in some centers, even though these advances offer benefit to the patient and render diagnostic precision possible to the neuroradiologist. It would seem that a balanced course should be encouraged. A procedure as modern as cerebral arteriography must progress by evolution, an evolution tempered with the knowledge that all new methods are not necessarily improvements, although some may offer genuine progress in safety and technique.

The earliest methods of cerebral arteriography entailed surgical exposure of the vessels. With the development of percutaneous puncture of the vessels, exposure of the carotid arteries was no longer necessary. Next came percutaneous direct puncture of the vertebral arteries which was technically difficult, unpredictable and hazardous to patients with vascular disease. A fortunate advance, indeed, was puncture of the brachial artery by cut-down or percutaneous methods and retrograde injection through a catheter or cannula needle. The modification of the Seldinger technique for brachial arteriography, with percutaneous catheterization, rendered surgical incision and direct exposure of the brachial arteries obsolescent. More recent advances in brachial arteriography, particularly with the introduction of the Rochester needle,* have rendered direct exposure of the vessel and the use of the cannula obsolete. Nevertheless, incision and exposure of the vessel has its advocates, although arteriograms obtained following direct exposure of the brachial artery are in no way superior to those obtained by the newer percutaneous techniques. Indeed, the patient is subjected to unnecessary surgery and the danger of ligation of the artery.

Percutaneous catheterization of the brachial arteries by the Seldinger technique continues to have a large following and appears to be a relatively safe procedure, although occasional thrombosis or embolism of the subclavian, axillary or brachial artery may occur and broken guide wires have been reported. It has also been apparent for several years, that equally good visualization of the subclavian and vertebral arteries may be obtained by injecting contrast material through an 18 gauge spinal needle, inserted percutaneously into the brachial artery. It is obviously simpler to place a spinal needle in the vessel in the cubital fossa, than to use the Seldinger technique and thread a catheter into the subclavian artery. In contrast, it is safer to inject through a catheter into a vessel, than through a needle. A compromise lies in the use of the Rochester needle, which permits the rapid introduction of a $4\frac{1}{2}$ inch polyethylene catheter into the brachial artery by one percutaneous puncture in the cubital fossa, the needle then being withdrawn. With the use of a pressure injector on the right side, it is possible to visualize the right subclavian, the right vertebral, the innominate and the right carotid arteries in all cases, particularly if seriograms are taken with a Schönader or Sanchez-Perez cassette changer. This allows the elimination of direct puncture of the right carotid artery, except in those very rare instances where the right carotid arises as a separate vessel from the left side of the aortic arch.

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* The Rochester needle is a device where a thin plastic catheter sheathes the needle for percutaneous puncture of the vessel and, after entry into the vessel, the needle may be discarded and the catheter advanced.

The problem of the elimination of direct left carotid artery puncture has proved to be more difficult. Some 20 per cent of cases will show satisfactory visualization of the left common carotid artery, using the right brachial technique with pressure injection through a Rochester catheter. Whether or not the left carotid artery is visualized does not seem to be a question of the pressure of injection, but probably depends upon anatomic variations in site of origin of the left common carotid artery from the aortic arch. Even with good filling of the left common carotid artery, the filling of the intracranial vessels may be poor or inadequate, or there is lack of detail due to superimposition of the vessels of the right hemisphere in the lateral view. With the routine use of seriograms and the introduction of rapid subtraction techniques, made possible by two television cameras, improved detail is possible, when the bolus enters the left common carotid artery. Unfortunately, in 80 per cent of cases the contrast material does not enter the vessel.

The major complications in cerebral arteriography are usually the direct result of percutaneous puncture of the left carotid artery and, since it supplies the dominant hemisphere in the great majority of people, neurologic deficits are often tragic. Subintimal or sheath injections are always a danger using an 18 gauge spinal needle, particularly if an attempt is made to advance the needle cranially after puncturing the vessel. The 18 gauge Cournand needle has been found to be a better instrument for direct carotid arteriography than the 18 gauge spinal needle, since the inner needle on the Cournand is removed after insertion into the vessel, leaving a metal cannula in the lumen. This cannula can be advanced without damaging the endothelium or entering the vessel wall.

When cerebrovascular disease is considered, it must be recognized that arteriography of the left carotid system, following direct puncture of the vessel, does not allow visualization of the common carotid artery, proximal to the Cournand cannula, down to its origin from the aortic arch. In any study of the four vessels of the neck and the aortic arch, using bilateral brachial arteriography and direct left carotid arteriography, this portion of the left common carotid artery will usually be the only part of the system which is not visualized. In those cases where arteriography of the aortic arch and the neck vessels is the major purpose of the study (particularly cases of cerebrovascular disease where surgical reconstruction is to be considered), an aortic arch arteriogram obtained by injection of contrast material into the ascending aorta via a teflon catheter is an excellent screening method. The catheter is placed in the femoral artery by the Seldinger technique and advanced in retrograde fashion into the ascending aorta. Pressure injection, depositing a large bolus (60 cc.) of contrast material in a short period of time and the use of a cassette changer for seriography, is again a necessity. This is a safe method of screening patients with cerebral vascular disease who are believed to have major stenotic lesions in the great vessels at the arch or the cervical vessels which may be amenable to surgical removal. Should more detailed views of the vessels be necessary after this screening method, brachial arteriography or direct left carotid arteriography, as already described, may be selected.

The wise application and selection of these methods make cerebral arteriography a relatively safe procedure, but, in order to reduce complications to a minimum, the patient must be adequately prepared for the study. In patients with cerebrovascular disease, arteriography should be the last procedure performed in a work-up which includes evaluation of cardiac function, hypertension, thyroid status, hyperuricemia, diabetes mellitus, hypercholesterolemia and hypoadrenalism secondary to pituitary insufficiency. Arteriosclerosis is the end product of many factors and appears to be enhanced by many associated diseases, some of which are amenable to treatment, and it is well to be aware of deficits in other systems before subjecting a patient with cerebral vascular disease to arteriography. Some patients with cerebral vascular disease may be extremely intolerant to stress, presumably due to poor adrenal function, and should be subjected to the least traumatic methods of arteriography. There should also be a constant alert for hypotensive episodes and a willingness to stop the procedure if they occur. In such cases, coverage with steroids during the procedures may be necessary.

In the future development of cerebral arteriography, the search for newer and safer techniques will be continued; an obvious goal is elimination of direct left carotid arteriography. The development of teflon catheters for retrograde brachial and femoral aortography, which have flexible tips that can be curved by manual control externally and introduced selectively into the vessels arising from the aortic arch, may be a solution to this problem and would certainly seem to be the next step. It is conceivable that with better contrast media and subtraction techniques the dream of intravenous cerebral arteriography may one day be a reality.

It is apparent that there have been major advances in cerebral arteriography, making it a safer and more definitive procedure. Further progress will be made, but, in the meantime, there is an obvious need for education of the physician in the wider adoption of these more modern techniques. Such techniques are well established in the specialty journals and, although the average arteriographer may find these new methods difficult to master at first in comparison to his well established routine, the advantages are usually readily appreciated, once this has been accomplished.

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NATIONAL COUNCIL ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

As announced in the September issue of the Journal, formation of the National Council on Radiation Protection and Measurements (NCRP), a non-profit corporation, was enacted by an Act of Congress (Public Law 88-376), which was signed by the President on July 14, 1964. The Council was brought into being at its organizational meeting held in Washington on August 3, 1964.

One of the principal objects of the Council will be to collect, analyze, develop and disseminate scientific information and recommendations about protection against radiation and about radiation measurement.

In this connection the Council will take over and continue the work previously carried out by the National Committee on Radiation Protection and Measurements, an association of scientists, which has for the last 35 years provided the basic material on which radiation protection activities in the United States are founded. The National Committee, established in 1929, has promulgated 31 Handbooks on radiation protection and measurements published by the National Bureau of Standards (NBS). The increasing importance of radiation protection efforts in the United States led the members of the Committee to seek formal status via a federal charter.

The significance of the work of these scientists and the need for the establishment of a formal organization received recognition when Senator John O. Pastore, Chairman of the Joint Committee on Atomic Energy, and Representative Chet Holifield, Vice-Chairman of the Joint Committee, introduced in the Senate and House of Representatives identical bills to charter the National Council. Testifying in support of the bills before Subcommittee 4 of the House Judiciary Committee in 1963, Congressman Holifield said: "As past Chairman and currently Vice-Chairman of the Joint Committee on Atomic Energy, and as one who has been a member of the Committee from its inception extending over 16 years, I have had many opportunities to evaluate the work of the NCRP. My belief in the soundness of the organization is the result of numerous studies, investigations and hearings conducted by the Joint Committee over the years, covering such matters as fallout from nuclear weapons testing; radioactive waste disposal; employee hazards and workmen's compensation; Federal-State relations in the control of Atomic Energy; and the development and application of radiation protection standards. In general, I think it can be said that the agencies of government rely heavily on the informed data and opinions of the NCRP."

Representative Holifield's support was important in obtaining final passage of the bills that created the new Council.

At its organizational meeting, the Council determined to continue the work begun by the previous Committee and took the initial steps necessary to form a viable organization capable of carrying out the purposes set forth in its charter.

Dr. Lauriston S. Taylor, a leader in radiation protection activities for three decades, was elected President of the Council; Dr. Shields Warren, an international authority in radiation pathology, was elected Vice-President. The Council also elected a 53-man Board of Directors, an Executive Committee and a Finance Committee. The Council plans to open an office in Washington in the near future and expects to hire a full-time Executive Director at that time.

Directors elected to the Board are: C. M. Barnes, Yamoto CMR, APO 323, Box 6119, San Francisco, Calif.; E. C. Barnes, Westinghouse Electric Corporation, Pittsburgh, Pa.; Victor P. Bond, Division of Microbiology, Brookhaven National Laboratory, Upton, L. I.,

N. Y.; C. B. Braestrup, Physics Laboratory, Francis Delafield Hospital, New York, N. Y.; James T. Brennan, Armed Forces Radiobiology Research Institute, National Naval Medical Center, Bethesda, Md.; Loy T. Brown, Medical Corps, Department of Radiology, U. S. Naval Hospital, Bethesda, Md.; Reynold F. Brown, University of California School of Medicine, San Francisco, Calif.; Frank R. Bruce, Director of Radiation Safety and Control, Oak Ridge National Laboratory, Oak Ridge, Tenn.; John C. Bugher, Biomedical Building, Caparra Heights Station, San Juan, Puerto Rico; D. R. Chadwick, Division of Radiological Health, Public Health Service, Department of Health, Education and Welfare, Washington, D. C.; Richard H. Chamberlain, University of Pennsylvania Hospital, Philadelphia, Pa.; James F. Crow, Department of Medical Genetics, University of Wisconsin, Madison, Wis.; R. L. Doan, Reactor Licensing Division, U. S. Atomic Energy Commission, Tucson, Ariz.; Charles L. Dunham, Division of Operational Safety, U. S. Atomic Energy Commission, Washington, D. C.; T. C. Evans, College of Medicine, State University of Iowa, Iowa City, Ia.; Everett G. Fuller, High Energy Radiation, Radiation Physics Division, National Bureau of Standards, Washington, D. C.; Robert O. Gorson, Department of Radiology, Jefferson Medical Center, Philadelphia, Pa.; J. W. Healy, General Electric Company, Schenectady, N. Y.; Paul C. Hodges, Visiting Professor of Radiology, Department of Radiology, University of Florida, Gainesville, Fla.; A. R. Keene, Hanford Atomic Products Operation, General Electric Company, Richland, Wash.; Morris Kleinfeld, Division of Industrial Hygiene, N. Y. State Department of Labor, New York, N. Y.; H. W. Koch, Chief, Radiation Physics Division, National Bureau of Standards, Washington, D. C.; John Laughlin, Department of Medical Physics, Memorial Hospital, New York, N. Y.; David I. Livermore, Headquarters, USAF (AFMSPA), Office of the Surgeon General, Washington, D. C.; George V. LeRoy, Billings Hospital, The University of Chicago, Chicago, Ill.; W. B. Mann, Chief, Radioactivity Section, National Bureau of Standards, Washington, D. C.; W. A. McAdams, Industry Standards Service, General Electric Company, Schenectady, N. Y.; G. W. Morgan, Division Civilian Applications, U. S. Atomic Energy Commission, Washington, D. C.; Karl Z. Morgan, Director, Health Physics Division, Oak Ridge National Laboratory, Oak Ridge, Tenn.; Russell H. Morgan, Department of Radiology, Johns Hopkins University, Baltimore, Md.; H. J. Muller, City of Hope Hospital, Duarte, Los Angeles, Calif.; R. J. Nelsen, Route 2, Great Falls Road, Rockville, Md.; Robert R. Newell, 310 Arballo Drive, San Francisco, Calif.; W. D. Norwood, Hanford Atomic Products Operation, General Electric Company, Richland, Wash.; Herbert M. Parker, Hanford Atomic Products Operation, General Electric Company, Richland, Wash.; Clinton Powell, 6607 Lone Oak Drive, Bethesda, Md.; Edith H. Quimby, Department of Radiology, Columbia University, New York, N. Y.; Robert Robbins, Department of Radiology, Temple University Hospital, Philadelphia, Pa.; Harald H. Rossi, Department of Radiology, Columbia University, New York, N. Y.; Eugene L. Saenger, Radioisotope Laboratory, Cincinnati General Hospital, Cincinnati, Ohio; Thomas L. Shipman, University of California, Los Alamos Scientific Laboratory, Los Alamos, N. M.; F. J. Shore, Department of Physics, Queens College, Flushing, N. Y.; Curt Stern, Department of Zoology and Genetics, University of California, Berkeley, Calif.; J. H. Sterner, Medical Director, Eastman Kodak Company, Rochester, N. Y.; Robert S. Stone, Department of Radiology, University of California Medical Center, San Francisco, Calif.; Lauriston S. Taylor, Associate Director, National Bureau of Standards, Washington, D. C.; E. Dale Trout, Radiation Center, Oregon State University, Corvallis, Ore.; Bernard F. Trum, Director, Animal Care Center, Harvard Medical School, Boston, Mass.; Shields Warren, New England Deaconness Hospital, Boston, Mass.; Edward W. Webster, Massachusetts General Hospital, Boston, Mass.; Forrest Western, Director, Division of Safety Standards, U. S. Atomic Energy Commission, Washington, D. C.; Edwin G. Williams, Division of Radiological and Occupational Health, Florida State Board of Health, Jacksonville, Fla.; Harold O. Wyckoff, Radiation Physics Division, National Bureau of Standards, Washington, D. C.

For additional information please contact W. R. Ney, Secretary, National Bureau of Standards, Washington, D. C. 20234.

THE ELEVENTH INTERNATIONAL CONGRESS OF RADIOLOGY

President-Elect Luigi Turano, on behalf of the Radiologists of Italy, extends a cordial invitation to all members of the National Societies of Radiology, Radiobiologists, Physicists and Allied Scientists and their families to attend The Eleventh International Congress of Radiology, which will be held in Rome, September 22–28, 1965, under the patronage of the President of the Republic of Italy, His Excellency, Prof. Antonio Segni.

"We Italians," says President-Elect Turano, "greatly appreciate the honor conferred upon us by our colleagues from so many nations, when the International Committee, gathered in Montreal on August 25, 1962, accepted the Italian offer and designated Rome as the place for the next Congress in 1965. We are aware of our responsibility to prove worthy of this honor and to ensure that this Congress will come up to the high level of previous Congresses. My fellow workers and I are doing, and will do our utmost to organize as well as possible this imposing scientific manifestation of our great discipline, all members of which will be warmly welcome."

The Sessions of the Congress will be held in the Congress House EUR (Esposizione Universale di Roma) where the Scientific and Technical Exhibition will also be located. The EUR is connected with the square of the Stazione Termini of the Metropolitan Railway and with other central areas of Rome by numerous urban transport services; it can also easily be reached by private cars for which ample parking space is available. Shops and restaurants can be found in the immediate vicinity of the Congress House.

The Secretariat of the Congress will be open in the Congress House from Monday, September 20, 1965 on.

The schedule of Session Meetings is as follows;

Monday, September 20: First session of the International Executive Commission.

Tuesday, September 21: morning—Session of the Council of the I.S.R.; afternoon—Session of the International Committee; evening—President's Reception for Members.

Wednesday, September 22: morning—Opening Ceremony of the Eleventh Congress, the Scientific Exhibition, and the Technical Exhibition; afternoon—Beginning of the Sessions of the Congress.

Thursday through Tuesday, September 23–28: Continuation of the Sessions of the Congress (except Sunday, September 26).

Thursday, September 23: Meeting of the General Assembly of the I.S.R.

Tuesday, September 28: afternoon—Closing ceremony of the Congress, of the Scientific Exhibition, and of the Technical Exhibition.

Receptions and social events will be organized for the days of the Congress; a time table will be announced in the final program booklet.

The papers to be presented to the Congress will be divided into invited papers and proffered papers. The time table will allow for approximately 225 invited papers, in the following divisions: Radiodiagnosis, Radiotherapy, Nuclear Medicine, Radiobiology, Radiological Physics and Techniques. A special session will be devoted to the discussion of teaching, and organizational and medico-legal problems connected with the various branches of Radiology. The invited papers will be presented in the Aula Magna and in the two main subsidiary halls. Other rooms will be available at the same time for the presentation of the proffered papers.

In selecting papers, the Scientific Committee of the Congress will give preference to new scientific and technical developments since the Tenth International Congress. Those interested in presenting papers should write to President-Elect Luigi Turano, Via Reno 21, Roma 1, Italy. The closing date for submission of summaries of scientific papers, as well as for applications of space for Scientific Exhibits and requests for hotel reservations, is December 31, 1964. The closing date for applications of space for the Technical Exhibition was September 30, 1964.

Preliminary programs and application blanks for membership in The Eleventh International Congress of Radiology have been mailed recently. The closing date for enrollment of members of all categories at normal fee and for submission of biographic data and photographs for the Book of Members is December 31, 1964. Physicians who are not members of national radiological organizations and wish to attend the Congress must be sponsored by a national radiological society.

The American Roentgen Ray Society at its Annual Meeting in October, 1963 elected Dr. James C. Cook of Detroit as its Delegate, and Dr. Melvin M. Figley of Seattle as its Alternate Delegate to the Congress. Dr. Earl E. Barth of Chicago is Chairman of the U. S. Delegation to the Congress.

The Board of Chancellors of the American College of Radiology has empowered Dr. Barth to sponsor, in the name of the College, physicians and others qualified to attend the Congress. Application blanks for membership may be obtained by writing to Dr. Earl E. Barth, Chairman of the U. S. Delegation to The Eleventh International College of Radiology, 20 North Wacker Drive, Chicago 6, Ill.

Rome has an international airport "Leonardo da Vinci" of Fiumicino, where the planes of Alitalia and of the other principal international airlines land. The air terminus is at the Central Station (Stazione Termini), which is also the only railroad station for the principal international railway lines. The nearest port for international shipping lines is Naples. The new "Autostrada del Sole" connects Naples with Rome, Florence, Bologna and Milan. A network of state highways connects Rome with the other principal cities of Italy.

Those attending the Congress are free to book their transportation with any agent of their choice. It is recommended, however, to book as soon as possible, because the tourist season is at its height in September.

All correspondence concerning hotel accommodation, etc., should be addressed to Wagons-Lits-Cook, Via Nizza 128, Rome (1), Italy.

After the closure of the Congress, a number of visits will be organized to university institutions, hospitals and research centers and these also will be listed in the final program booklet.

Rome, the eternal city, with its "seven hills," the ancient "Lapis Niger" in the Roman Forum, the splendid Colosseum, the incomparable Basilica of St. Peter and apostolic palaces, magnificent monuments, archeologic treasures, and exquisitely "beautiful fountains" epitomizes the history of mankind over a span of 27 centuries of cultural growth and artistic development. Countless immortal works of art by famous masters are matched only by the age-old pine trees with grandiose views of the luxurious country around Rome. Modern autostradas provide easy accessibility to many attractive tourist centers for which Italy is renown.

The American College of Radiology, through its Executive Director, announces that it plans to arrange group travel in connection with this Congress. Those interested please write to the College office, 20 North Wacker Drive, Chicago 6, Ill. for further information.

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NEWS ITEMS

ACR FOUNDATION MUSEUM

The Executive Committee of the American College of Radiology Foundation during its meeting in San Francisco in June, 1964 approved the following: (1) The appointment of Mr. Robert T. Morrison of the Eastman House as Curator of the ACR Foundation Museum; and (2) the empowering of the Museum Committee and Mr. Morrison to initiate and carry out an intensive search for the location, identification, authentication and registration of all possible pieces of equipment and memorabilia, including letters, books, notebooks, documents, doodling papers, personal miscellany, photographs, magazines, reprints, business documents, catalogs, lecture notices, manuscripts, x-rays, cartoons, society records, teaching material, etc.

The procedure will be to first, write a letter to Mr. Robert T. Morrison, 900 East Ave., Rochester, N. Y. 14607, describing the item(s). He will then have the material studied and checked against a master procurement list and against other requirements. When found suitable, the Committee of Museum will request the donor's signature on an official transfer form. No item will be accepted unless it is donated unconditionally. Tags made of durable material will be affixed to the objects. On these will be imprinted: Property of the American College of Radiology Foundation; name of the donor; acquisition number; date; and description of the object; as well as other pertinent data.

Mr. Morrison will direct the shipment to Eastman House or to any one of many depositories made available for these items by the Picker Corporation, Eastman Kodak, and many others, ensuring that the material is properly identified and that it is being held in temporary storage for the ACR Foundation.

If desirable or more convenient, the Chairman or any member of the Museum Committee may be contacted directly. M. H. Poppel, M.D., Chairman, Lloyd E. Hawes, M.D., Andre J. Bruwer, M.D., John M. Dennis, M.D., E. R. N. Grigg, M.D., Lewis E. Etter, M.D., Loy T. Brown, M.D., Ralph Voorhees, D.D.S., Ivor Bennett and R. A. Olden.

SEVENTEENTH ANNUAL MIDWINTER RADIOLOGICAL CONFERENCE

The Seventeenth Annual Midwinter Radiological Conference, sponsored by the Los Angeles Radiological Society, will be held at the Biltmore Hotel, Los Angeles, California, on Saturday, January 30 and Sunday, January 31, 1965.

An outstanding program has been arranged featuring the following speakers: Professor Olle Olsson, Lund, Sweden; Donald L. McRae, M.D., Montreal, Canada; Harry Z. Mellins, M.D., New York, N. Y.; Vincent P. Collins, M.D., Houston, Tex.; and Dr. Eric Samuel, Edinburgh, Scotland.

Hotel reservations may be made directly by contacting the Convention Manager, Biltmore Hotel, Los Angeles, California.

Conference Reservations should be made through John L. Gwinn, M.D., Secretary-Treasurer, Childrens Hospital, 4614 Sunset Blvd., Los Angeles 27, California.

W. EDWARD CHAMBERLAIN LECTURE OF 1964

The W. Edward Chamberlain Lecture of 1964 will be given by Dr. Nils-Magnus Ohlsson of the University of Lund, Malmo, Sweden, on November 4, 1964 at 4:00 P.M. in the Auditorium of the Temple University School of Medicine, Philadelphia, Pennsylvania. The title of Dr. Ohlsson's Lecture is *"Current" Events in the Aorta: Experimental Cineradiographic Studies of Flow Patterns.*

BOOKS RECEIVED

Give and Take: The Development of Tissue Transplantation. By Francis D. Moore, M.D., Moseley Professor of Surgery, Harvard Medical School, Surgeon-in-Chief, Peter Bent Brigham Hospital, Boston, Mass. Cloth. Pp. 182, with some illustrations. Price $5.50. W. B. Saunders Company, 218 West Washington Square, Philadelphia, 1964.

Early Treatment of Facial Injuries. By Thomas John Zaydon, M.D., F.A.C.S., Clinical Assistant Professor of Surgery, Plastic Surgery Service, Department of Surgery, University of Miami School of Medicine; Chief, Plastic Surgery Service of St. Francis Hospital, Cedars of Lebanon Hospital, North Miami General Hospital, Miami, Fla; and James Barrett Brown, M.D., F.A.C.S., Professor of Clinical Surgery, Washington University School of Medicine; Professor of Maxillofacial Surgery, Washington University School of Dentistry, St. Louis, Mo.; Senior Civilian Consultant in Plastic and Maxillofacial Surgery to the Surgeon General; Special Consulting Plastic Surgeon to United States Veterans Administration; Senior Consultant in Plastic and Maxillofacial Surgery and Burns, United States Army. Cloth. Pp. 258, with 441 illustrations. Price, $15.00. Lea & Febiger, Washington Square, Philadelphia, 1964.

Primary Intramedullary Tumors of the Spinal Cord and Filum Terminale. By Johan L. Slooff, M.D., Special Appointee of the Mayo Foundation, Section of Pathologic Anatomy, Mayo Clinic, Rochester, Minn., 1959–1961; Pathologist, Katholieke Universiteit, Nijmegen, The Netherlands; James W. Kernohan, M.D., M.A., Emeritus Consultant, Section of Pathologic Anatomy, Mayo Clinic; Emeritus Professor of Pathology, Mayo Foundation, Graduate School, University of Minnesota, Rochester, Minn.; and Collin S. MacCarty, M.D., M.S., Head, Section of Neurologic Surgery, Mayo Clinic; Professor of Neurologic Surgery, Mayo Foundation, Graduate School, University of Minnesota, Rochester, Minn. Cloth. Pp. 255, with many illustrations. Price, $13.50. W. B. Saunders Company, 218 West Washington Square, Philadelphia, 1964.

Introduction to Molecular Biology. By G. H. Haggis, M.A., Ph.D., Editor, Lecturer in the Department of Physiology, University of Edinburgh; D. Michie, M.A., D.Phil., Reader in the Department of Surgical Science, University of Edinburgh; A. R. Muir, M.D., Reader in the Department of Anatomy, University of Edinburgh; K. B. Roberts, M. A., D.Phil., M.B., B.S., Senior Lecturer in Department of Physiology, London Hospital Medical College, London; and P. M. B. Walker, B.A., Ph.D., Reader in the Department of Zoology, University of Edinburgh. Cloth. Pp. 401, with many illustrations. Price $7.50. John Wiley & Sons, Inc., Publishers, 605 Third Avenue, New York 16, 1964.

Scheuermann's Juvenile Kyphosis: Clinical Appearances, Radiography, Aetiology, and Prognosis. By K. Harry Sørensen. Paper. Pp. 273, with some illustrations and many tables. Munksgaard, Copenhagen S, Denmark, 1964.

Practical Procedures in Diagnostic Radiology. By H. M. Saxton, M.B., M.R.C.P., F.F.R., D.M.R.D., Consultant Radiologist, Guy's Hospital, London; and Basil Strickland, M.B., M.R.C.P., D.C.H., F.F.R., D.M.R.D., Consultant Radiologist, Westminster Hospital, London; Consultant Radiologist, Brompton Hospital, London. Cloth. Pp. 275, with some illustrations. Price, $8.75. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, 1964.

Radiologic Clinics of North America. Symposium on Radiology and the Skeletal System. Volume II. Number 2. Guest Editor, Gwilym S. Lodwick, M.D. Cloth. Pp. 374, with many illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, 1964.

Radiologia Infortunistica Della Colonna Vertebrale: Patologia Traumatica e Medicina Legale. By Lorenzo Giuntoli, and Bruno Guareschi, Istituto di Radiologia dell'Università di Milano, Italy. Cloth. Pp. 436, with 272 illustrations. Price, Lire 12.000. L. Cappelli, Via Farini 6, Bologna, Italy, 1963.

ABSTRACTS OF RADIOLOGICAL LITERATURE

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Neck and Chest

Holt, E. P., Jr.: Fallacy of cervical discography: report of 50 cases in normal subjects.. 959
Adler, D. C., Haverback, B. J., and Meyers, H. I.: Cineradiography of esophageal varices 959
Gandini, D., and Petrocini, S.: Clinical radiologic features of infantile and adolescent adeno-broncho-parenchymal lesions following primary pulmonary tuberculous infection. 959
Dubowitz, L. M. S., and Dubowitz, V.: Acute dermatomyositis presenting with pulmonary manifestations 960
Kaye, J., and Murray, J. F.: Histoplasmosis: the radiological features of the benign pulmonary form in South Africa 960
Ariel, I. M., and Lear, P.: The diagnosis of pulmonary embolism by radiating microspheres 960
Roland, A. S.: The syndrome of benign thymoma and primary aregenerative anemia: an analysis of forty-three cases 960
Forfar, J. O., *et al.*: Endocardial fibroelastosis 961
Ferencz, C.: Visceral heterotaxy with malformed heart: similar findings in a child and a puppy 961

Abdomen

Evans, J. A.: Specialized roentgen diagnostic technics in the investigation of abdominal disease: Annual Oration in memory of Clarence Elton Hufford, 1893–1958 961
Birch, J. R., Shea, J., and Currie, D. J.: Transhepatic cholangiography 962
Myers, R. N. *et al.*: Cinecholangiography as an aid in the interpretation of T-tube cholangiogram deformities 962
Pearson, J. B.: Torsion of the spleen associated with congenital absence of the left kidney 963

Genitourinary System

Heetderks, D. R., Jr. *et al.*: An improved method of excretory urography 963
Fam, A.: The problem of bilharzial ureter 963
Valvassori, G. E., and Pierce, R. H.: Osteosclerosis in chronic uremia 964
Dent, C. E., and Friedman, M.: Hypercalcuric rickets associated with renal tubular damage 964
Kreuzer, H., and Sturm, A., Jr.: Pseudopheochromocytoma 965

Skeletal System

Pillay, V. K.: Ophthalmo-mandibulo-melic dysplasia: an hereditary syndrome 965
Wee, L. K., and Pillay, V. K.: Hereditary cleido—cranial dysostosis: with a note on the anomaly of cementum 965
Pollard, J. J., and New, P. F. J.: Hereditary cutaneomandibular polyoncosis: a syndrome of myriad basal-cell nevi of the skin, mandibular cysts, and inconstant skeletal anomalies 965
Shuler, S. E.: Pycnodysostosis 966
Plastina, U., Pandolfo, G., and Crosca, A.: Osteopathy in hypophosphatemia: clinical and radiologic findings 966
Paul, S. S. *et al.*: A case of chondrodystrophia calcificans congenita 966
Chawla, S., and Bery, K.: Iliac horns with arthrodysplasia and dystrophy of the nails—Fong's lesion: (review of the literature and report of a family) 967
Joyce, J. J., III, and Grant, B. D.: Early diagnosis of slipping of the upper femoral epiphysis 967
Lichtenstein, L., and Sawyer, W. R.: Benign osteoblastoma: further observations and report of twenty additional cases 967
Jewell, J. H., and Bush, L. F.: "Benign" giant-cell tumor of bone with a solitary pulmonary metastasis: a case report 968
Dowling, E. A.: Mesenchymal chondrosarcoma 968
Phelan, J. T., and Cabrera, A.: Chondrosarcoma of bone 968
Black, S. P. W., and Keats, T. E.: Generalized osteosclerosis secondary to metastatic medulloblastoma of the cerebellum 969
Beller, H. E., and Kirsh, D.: Spondylolysis and spondylolisthesis following low back fusions 969
Rhangos, W. C., and Chick, E. W.: Mycotic infections of bone 969

Blood and Lymph System

Lampe, W. T., II.: A severe reaction to angioconray 970

Meyer, M. W., and Read, R. C.: Red cell aggregation from concentrated saline and angiographic media 970

Whitesel, J. A.: Lymphography: its place in urology 971

Brody, H. S., Helff, J. R., and Bujdoso, L.: Lymphangiography: an aid in urological surgery 971

Ortiz, F., Walzak, M. P., and Marshall, V. F.: Chyluria: lymphatic-urinary fistula demonstrated by lymphangiography 972

Koehler, P. R. *et al.:* Body distribution of ethiodol following lymphangiography 972

General

McCort, J., and Vaudagna, J.: Visceral injuries in battered children 972

Goidanich, I. F., and Lenzi, L.: Morquio-Ullrich disease: a new mucopolysaccharidosis 973

Zarnstorff, W. C., and Rowe, G. G.: Stereoscopic fluoroscopy and stereographic cineangiocardiography 973

Bohatirchuk, F.: Use of x-rays in the detection of calcium in biological tissues at microlevel 973

RADIATION THERAPY

Walter, L. *et al.:* Assessment of response of cervical cancers to irradiation by routine histological methods 974

Rimondi, C., and Mazzanti, G.: Complications and after effects of high energy radiation therapy of pulmonary neoplasms 974

Reeves, R. J., and Worde, B. T.: Preliminary observations in cesium 137 teletherapy 975

Duggan, H. E.: Effect of x-ray therapy on patients with Peyronie's disease 975

Duggan, H. E., and Weijer, D. L.: Eustachian tube obstruction and radiation therapy 975

Parker, R. G. *et al.:* Radiation cataract in clinical therapeutic radiology 976

ROENTGEN DIAGNOSIS

Neck and Chest

Holt, Earl P., Jr. Fallacy of cervical discography: report of 50 cases in normal subjects. *J.A.M.A.*, June 1, 1964, *188*, 799–801. (Address: 3720 Washington Boulevard, St. Louis, Mo.)

This is a report of a cervical diskography study on 50 volunteers who were inmates of the Missouri State Prison. The subjects were between 21 and 50 years of age, with no history of cervical injuries and without a history of neck or arm pain.

Injection of 148 spaces in the 50 volunteers was made with 50 per cent hypaque sodium. The procedure caused intense pain regardless of the amount of injected material. There was extravasation in all but 10 per cent of the injected spaces.

The study was undertaken because no such project has been reported upon in normal individuals.

The author concludes that extravasation of the opaque material is without diagnostic significance. Also, that the claim of reproduction of pain by injection of the suspected disk space is fallacious. The volume of injectable material is unreliable as an indication of pathology.

Thus, since cervical diskography is a painful and expensive procedure without diagnostic value, its use is not considered to be contributory.

Reproductions of 4 roentgenograms and a chart of age groups, disks injected and extravasation pattern are included.—*John H. Harris, M.D.*

Adler, Denis C., Haverback, Bernard J., and Meyers, Harvey I. Cineradiography of esophageal varices. *J.A.M.A.*, July 13, 1964, *189*, 77–80. (Address: Dr. Adler, Suite 408, 2010 Wilshire Boulevard, Los Angeles 57, Calif.)

The diagnostic accuracy of cineradiography was measured utilizing a selected group of cirrhotic patients in whom the presence of varices was highly probable. The diagnosis of esophageal varices was correlated by endoscopy on the same group of patients and proved to be in agreement in 85 per cent of the patients studied. Disagreement in 15 per cent of the cases occurred in that group of patients with extremely small varices and in which minor changes were exhibited. This diagnostic method also proved valuable in studying the dynamic and morphologic changes which occur in the esophagus of the cirrhotic patient.

The authors studied 76 patients using an image amplifier which covered a field 22 cm. in diameter. A synchronization device energized the fluoroscopic tube only when the camera shutter was open, with filming being done at 7½ and 15 frames per second.

The examinations were performed with the patients in the prone oblique position, using thick barium paste. The Valsalva maneuver was routinely employed in an attempt to increase the intrathoracic and intra-abdominal venous pressure. This maneuver proved frequently valuable in providing better visualization of varices than was obtained during simple inspiration and expiration roentgenography.

The major diagnostic criteria for esophageal varices are the presence of nodular filling defects and widened, tortuous, mucosal folds. When these changes were present to a marked degree, no difficulty was encountered in demonstrating them on spot roentgenograms. However, with small varices and minor changes which are unimpressive on single roentgenograms, the importance of continuous filming becomes apparent. These same minor changes when shown on dozens of films assume definite pathologic significance.

Of the 76 patients studied, 67 were diagnosed as having varices cineradiographically. Three cases were inconclusive due to technical factors involving the studies. Correlation in 85 per cent of the cases with endoscopic findings was noted as to both the presence or absence of varices. Comparison with previous studies utilizing conventional roentgenologic method and esophagoscopy is mentioned.—*Donald S. Faust, M.D.*

Gandini, D., and Petrocini, S. Quadri clinico-radiologici di lesioni adeno-bronco-parenchimali della prima infezione tubercolare polmonare e dei suoi postumi, nell'infanzia e nell'adolescenza. (Clinical radiologic features of infantile and adolescent adeno-broncho-parenchymal lesions following primary pulmonary tuberculous infection.) *Radiol. med.*, April, 1964, *50*, 305–341. (Address: Dott. Prof. Dario Gandini, Via Torino, 35, Biella (Vercelli), Italy.)

The authors present 10 cases studied by conventional roentgenography, laminagraphy, bronchography and broncho-laminagraphy.

They draw the following conclusions:

The tubercular infection in the pediatric age, thanks to modern therapeutic methods, has a relatively benign course, and mortality is reduced to a minimum.

The primary infection evolves rapidly and favorably when it is manifested as a primary complex or as a simple tracheo-bronchial adenopathy.

It is accompanied frequently by secondary parenchymal involvement; *i.e.*, allergic exudative phenomena, emphysema and atelectasis.

The evolution of the first infection is as a rule benign. Sometimes, it may produce irreversible broncho-parenchymal lesions which are characterized by hemoptotic, circumscribed bronchitic, stenotic and bronchiectatic manifestations, occasionally complicated by nonspecific infections.—*Anthony A. Blasi, M.D.*

Dubowitz, Lilly M. S., and Dubowitz, Victor. Acute dermatomyositis presenting with pulmonary manifestations. *Arch. Dis. Childhood,* June, 1964, *39,* 293–296. (From: The Children's Hospital, Sheffield, and Department of Child Health, University of Sheffield, Sheffield, England.)

Dermatomyositis has been defined as an acute, subacute or chronic disease of unknown etiology characterized by edema, dermatitis, and a nonsuppurative inflammation of striated muscle.

The authors present the case of a 5 year old boy with clinical and roentgenologic features of miliary tuberculosis which subsequently proved to be dermatomyositis. The child also developed abdominal distension which was thought to be due to involvement of the musculature of the small bowel by the disease process.

The chest roentgenogram showed miliary shadows probably reflecting pulmonary infiltration due to dermatomyositis since the lungs cleared completely within 10 days of steroid therapy. Though pneumonitis is a well documented feature of various collagen diseases, there are very few reported cases in association with dermatomyositis.—*Francis P. Shea, M.D.*

Kaye, Josse, and Murray, J. F. Histoplasmosis: the radiological features of the benign pulmonary form in South Africa. *Med. Proc.,* May, 1964, *10,* 176–183. (From: Department of Radiology, University of the Witwatersrand and the Johannesburg General Hospital; and the South African Institute for Medical Research, Johannesburg, South Africa.)

The authors report on the roentgenologic features of the benign form of pulmonary histoplasmosis as observed in a study of the disease in South African speleologists. These features are based upon initial and follow-up roentgenographic examinations of 48 out of 62 known cases. The study includes 13 cases exhibiting miliary mottling scattered throughout one or both lungs; 8 cases in which the miliary mottling was associated with crayon lines, thick, coarse, linear shadows clearly distinguishable from branching blood vessels; 1 case showing only an irregular pneumonic consolidation; 2 cases showing nodular opacities larger than 4 mm. in diameter; and 2 cases exhibiting nodular opacities and a crayon line network. When present, crayon lines formed either a discrete or reticular pattern.

It is striking that none of the cases reported showed any evidence of coin lesions, hilar lymph node enlargement, cavitation or pneumothorax. Pulmonary calcification was also rarely encountered. The authors point out that only 16 per cent of the cases followed showed any evidence of miliary calcification and then only after a period of 3 to 9 years after the initial infection. The lack of these features tends to distinguish the South African variety of the disease from the strictly American variety of pulmonary histoplasmosis. The authors' conclusion stresses this fact.

Sixteen roentgenographic reproductions along with 2 case histories and a short historic review of the world literature on the subject of histoplasmosis are included in this text.—*Kenneth M. Nowicki, M.D.*

Ariel, Irving M., and Lear, Philip. The diagnosis of pulmonary embolism by radiating microspheres. *Radiology,* May, 1964, *82,* 876–878. (Address: 139 East 36th Street, New York 16, N. Y.)

The authors describe the technique and result of animal experimentation in which radioactive ceramic microspheres were employed to diagnose induced pulmonary emboli. Such microspheres (50±10 microns), when injected into peripheral veins, are trapped in the pulmonary arterioles where they remain indefinitely. Subsequent radioisotope procedures allow the demonstration of a circulatory block, represented by a "cold" region on the scan.

The animal studies were initially performed in rabbits after ligation of either a major pulmonary artery or a lobar artery. Microspheres labeled with the beta-emitter Yt^{90} were then injected after which the lungs were excised and studied by radioautography. Later, mongrel dogs were used wherein a thrombus was released from an iliac vein and then gamma-emitting radioactive microspheres were injected intravenously about 5 to 10 minutes later. After 15 minutes, the animals were sacrificed and the heart and lungs were removed and externally scanned. Of the various isotopes employed in the investigation by scanning, radiomercury (Hg^{203}) was found to be the most satisfactory.

The authors conclude that this technique, at least in animals, is feasible and safe since the amount of circulation compromised is negligible, representing less than 1/100th of the pulmonary circulation. The method of arriving at this figure is not presented, but it is stated that studies are in progress to ensure the safety of the procedure in man. Acknowledgment is made of the recently reported pulmonary scanning studies, in man, using I^{131} human serum albumin.—*E. B. Best, M.D.*

Roland, Arnold S. The syndrome of benign thymoma and primary aregenerative anemia: an analysis of forty-three cases. *Am. J. M. Sc.,* June, 1964, *247,* 719–731. (From: Department of Medicine, Hospital of the University of Pennsylvania, Philadelphia, Pa.)

There is an association between thymoma and the clinical syndromes of myasthenia gravis, Cushing's syndrome, agammaglobulinemia and primary aregenerative anemia. The last syndrome is thought to occur in 5–10 per cent of patients with thymoma while in adults it is probable that the syndrome of primary aregenerative anemia has an incidence of thymoma exceeding 50 per cent.

In reviewing 43 cases the following features were of note: the syndrome almost always occurred after the fourth decade, usually in the sixth; it was found twice as often in women as in men; the histology of the thymoma was uually a benign spindle cell variety in contrast to the more frequent thymoma composed of lymphocytes and epithelial cells.

Clinically these patients may present with symptoms of severe slowly progressive anemia. The anemia is normocytic and normochromic with low reticulocyte count and a marrow hypoplastic in erythroid elements. Depression of the myeloid and megakaryocytic elements may occur in association.

Therapy by thymectomy, steroids, testosterone or irradiation may be beneficial. Most of the patients who underwent remission had either surgical or radiation thymectomy in addition to steroids. No humoral cause for the anemia has been found.—*Fredrick S. Mishkin, M.D.*

FORFAR, J. O., MILLER, R. A., BAIN, A. D., and MACLEOD, W. Endocardial fibroelastosis. *Brit. M. J.*, July 4, 1964, *2*, 7–12. (Address: Dr. Forfar, Consultant Paediatrician, Western General Hospital, Edinburgh, Scotland.)

A comprehensive statistical study is made of 72 cases of endocardial fibroelastosis from 8,595 consecutive pediatric postmortem examinations between 1946–1962. Naked eye visualization of thick, opaque endocardium was the main diagnostic criterion used. Of 433 total cases of congenital heart disease, 17 per cent had endocardial fibroelastosis. The majority of the 72 cases had complicating lesions, most common being small left ventricle, aortic stenosis and coarct mitral stenosis. The incidence of these complicating lesions was greater than seen in the total population of congenital heart disease. The incidence of ventricular septal defect, atrial septal defect, and tetralogy of Fallot in endocardial fibroelastosis was significantly decreased. The association of lesions causing stagnation within cardiac chambers, as opposed to the diminished association of lesions causing rapid flow through cardiac chambers, suggests an acquired etiology. Supportive data, such as the absence of any cases of endocardial fibroelastosis in the 3,836 stillbirth necropsies, are further discussed.

The clinical symptoms, course, electrocardiographic and roentgenologic findings are reviewed. In 48 of the 52 patients studied roentgenographically (93 per cent) there was obvious cardiomegaly.

Suggested differences between this study and others are related to methods of case collection, failure to assess endocardial fibroelastosis against a background of congenital heart disease in general, and the vagaries of histologic diagnosis.—*Mark D. Reiss, M.D.*

FERENCZ, CHARLOTTE. Visceral heterotaxy with malformed heart: similar findings in a child and a puppy. *Anat. Rec.*, June, 1964, *149*, 299–307. (From: Department of Pediatrics, State University of New York at Buffalo, and the E. J. Meyer Memorial Hospital, Buffalo, N. Y.)

An 18 month old male with severe progressive cyanosis and dyspnea was examined roentgenographically and angiocardiographically. There was complete situs inversus with levocardia, transposition of the great vessels, hypoplasia of the anatomic left atrium and ventricle, mitral and pulmonary atresia, anomalous drainage of the pulmonary veins into the anatomic right atrium, and patent ductus arteriosus with narrowing at the pulmonary end. A subclavian-pulmonary anastomosis was performed with marked clinical improvement, and a near-normal state for 2 years.

A newborn puppy was examined at necropsy during the course of an unrelated study. There was complete thoracic situs inversus, including dextrocardia. The abdominal viscera were not examined. The remaining cardiac examination revealed anomalies of essentially identical nature to the child.

The association of identical anomalies in the human and canine species, as well as reports of similar conditions in the literature, suggests a specific syndrome. Classification of these complicated anomalies may lead to clues concerning the developmental deviation, and hence the etiology. A concluding plea is made for surgical help in other seemingly helpless conditions.—*Mark D. Reiss, M.D.*

ABDOMEN

EVANS, JOHN A. Specialized roentgen diagnostic technics in the investigation of abdominal disease: Annual Oration in memory of Clarence Elton Hufford, 1893–1958. *Radiology*, April, 1964, *82*, 579–594. (Address: 535 East 68th Street, New York, N. Y. 10021.)

Percutaneous cholangiography and preoperative visceral arteriography have proved to be extremely valuable diagnostic aids in patients with obscure or complex diseases of the abdominal organs.

Patients with jaundice frequently present a difficult diagnostic problem, and commonly employed methods of diagnosis fail to identify the cause of the jaundice. In such situations percutaneous transhepatic cholangiography permits differentiation between extrahepatic or obstructive and intrahepatic or parenchymal jaundice. The purposes for which percutaneous cholangiography is more useful than any of the other commonly employed diagnostic methods are:

1. To differentiate intrahepatic or parenchymal jaundice from extrahepatic or obstructive jaundice.
2. To determine the presence and number of common duct calculi.
3. To demonstrate intrinsic tumors of the bile ducts.
4. To disclose the presence of extrinsic tumors obstructing the common duct.
5. To detect the site and extent of stenosis of the bile ducts secondary to injury.
6. To discover intrahepatic calculi.
7. To disclose the presence and extent of sclerosing choledochitis.

The cholangiographic appearance of stone, stricture and carcinoma of the ampulla, common bile duct, or pancreas is frequently specific and diagnostic. A stone impacted in the common duct produces a deep concave meniscus with thin, acutely angled margins or corners. Carcinoma of the pancreas is usually associated with marked dilatation of the common bile duct. The obstructed end may be rounded, bulbous, tapered, or notched or may have a small nipple-like termination. Carcinoma of the ampulla causes dilatation of the common bile duct. There may be an uneven, ragged margin or a smooth, flat, shallow meniscus at the obstruction.

This examination carries a definite risk of serious complication, but the risk can be minimized by the use of narrow gauge needles and television monitoring. Because of the possible hazard, the procedure should not be attempted by inexperienced personnel or in the absence of immediately available surgical help. In 75 per cent of the author's 93 cases the procedure was considered successful.

Splenoportography by percutaneous injection of the spleen has definite value for preoperative study in candidates for portal decompression. In selected cases it may yield valuable information about pathologic conditions in the liver and pancreas. Puncture of the normal spleen, however, carries a considerably higher risk of complications than does puncture of the large fibrotic spleen associated with cirrhosis.

Operative intestinal arteriography has definite value to define regional vascular anatomy and to facilitate the surgical resection of gastrointestinal neoplasms. Selective catheterization of the major arterial systems can be done; it has been demonstrated that the celiac and mesenteric arteries can be catheterized and simultaneously opacified. This procedure can furnish valuable information in many diagnostic problems affecting the liver, pancreas, stomach and intestines. Other uses are: to detect occlusive lesions of the mesenteric arteries, arteriovenous fistulas of the intestine or other abdominal organs, and to determine viability of intestinal anastomosis. The author has performed 125 celiac and/or mesenteric arteriographic examinations without complications of any kind.

It is anticipated that, with more experience and attention to detail, diagnostic criteria will be refined, permitting earlier recognition of disease processes, especially in those abdominal organs which have hitherto been notoriously difficult to approach diagnostically.—*A. W. Sommer, M.D.*

Birch, John R., Shea, John, and Currie, Donald J. Transhepatic cholangiography. *Canad. M. A. J.*, June 27, 1964, *90*, 1442–1449. (From: Department of Surgery and Department of Radiology, St. Michael's Hospital, Toronto, Ontario, Canada.)

Transhepatic cholangiography is a means of roentgenographic visualization of the biliary tree. Its purpose is to diagnose and localize obstructing biliary lesions. The procedure is done by injecting contrast material by needle through the abdominal wall and liver into an intrahepatic bile duct.

Percutaneous transhepatic cholangiography has been most helpful in the preoperative evaluation of the poor risk patient who is jaundiced. Unnecessary operations can be prevented and correctable lesions can be diagnosed. The procedure can also be used in selected cases of hepatic enlargement when oral and intravenous cholangiography are unsuccessful. Transhepatic cholangiography has also been used during surgical procedures when localization of a biliary obstruction is desired.

Adequate cholangiograms are obtained in 80 per cent of the attempted cases. When the procedure is unsuccessful, hepatocellular disease is usually the cause of the jaundice.

In 5 per cent of collected cases, bile leakage with chemical peritonitis occurs as a complication of the procedure; 2 deaths have been reported from bile leak. One case of serious hemorrhage from the hepatic puncture site has been reported. The over-all death rate from percutaneous transhepatic cholangiography is low, however. Out of 418 cases collected from the literature, 2 deaths were reported.—*John A. Stryker, M.D.*

Myers, Richard N., Haupt, George J., Birkhead, Newton C., and Deaver, J. Montgomery. Cinecholangiography as an aid in the interpretation of T-tube cholangiogram deformities. *Surg., Gynec. & Obst.*, July, 1964, *119*, 47–50. (From: Surgical Service "A" and the Division of Research, Lankenau Hospital, Philadelphia, Pa.)

The presence of a common duct stone cannot always be excluded when apparent abnormalities of the bile duct system are demonstrated in the postoperative T-tube cholangiogram.

The authors utilized the technique of cinecholangiography in 12 cases in which persistent filling defects or abnormalities were visualized in multiple (2–5) postoperative cholangiograms. In each case it was demonstrated that the filling defect was due to an anatomic or physiologic mechanism rather than stone.

Three types of abnormality were observed. (1) In 6 of the cases there was a deformity of the distal portion of the common bile duct without any obstruction. This type of deformity on cinefluorography was intermittent and transient, and due to the action of the sphincter of Oddi in combination with duodenal peristalsis. (2) In 4 of the cases there were filling defects in the common and hepatic ducts. These were interpreted in the cinefluorographic studies to be either air bubbles or incomplete filling of radicles. (3) In 2 cases there was common duct deformity at the distal end with apparent obstruction. This was found to be associated with increase in tone and very brief or transient relaxations of the sphincter of Oddi.

Case histories and examples of each type of deformity are presented by the authors. None of the cases described had difficulty after removal of the T-tube with follow-up of from 6 to 18 months.

The authors suggest that postoperative cinecholangiography need not be used routinely but appears to be helpful in resolving the problem of the abnormal postoperative T-tube cholangiogram.—*James R. Knapp, M.D.*

Pearson, J. B. Torsion of the spleen associated with congenital absence of the left kidney. *Brit. J. Surg.*, May, 1964, *51*, 393–395. (From: Department of Surgery, University Hospital, Ibadan, Nigeria.)

Various reports of splenic torsion presenting as surgical abdominal problems have appeared in the literature. A rare case of splenic torsion and a congenitally absent left kidney diagnosed as a Wilms' tumor is herein reported.

A 1 year old male with a short history of fever, emesis, and an abdominal mass was examined. Intravenous pyelography revealed a normal functioning right kidney and a large nonexcreting soft tissue mass in the left flank extending into the whole lower abdomen. Survey roentgenograms of the chest and skeleton were normal. A Wilms' tumor was suspected. At operation a 110 gram spleen with its pedicle twisted two and one half times was found and splenectomy performed. No left kidney was present.

The absence of the lieno-renal ligament is suggested as predisposing to the splenic torsion.—*John T. Underberg, M.D.*

Genitourinary System

Heetderks, Dewey R., Jr., Rapp, Robert, Correa, Roy J., Jr., and Lapides, Jack. An improved method of excretory urography. *J. Urol.*, July, 1964, *92*, 75–77. (From: Department of Surgery, Section of Urology, University of Michigan Medical Center; and the Department of Radiology, Veterans Administration Hospital, Ann Arbor, Mich.)

This brief article describes an additional procedure which aids in obtaining good pyelo-ureterograms by the intravenous method. The addition to the routine technique which the authors recommend consists of placing the patient in the 15 degree Trendelenburg position and applying pressure over the lower ureters after the 5 minute urogram had been made. The lower half of the 14×17 inch cassette is covered by an appropriate piece of lead and the exposure is made of the contrast-filled renal pelves at the end of a 15 minute period. The roentgen-ray tube is then moved to the lower half of the film. The lead protecting shield is moved to the upper half of the cassette and another single exposure is made after the release of pressure. At this time, the lower ureters are well filled.

It is felt that by this procedure such satisfactory pyelo-ureterograms are obtained that one can reduce the number of retrograde pyelograms necessary.

Four illustrations accompany this brief report.—*George W. Chamberlin, M.D.*

Fam, Aziz. The problem of bilharzial ureter. *Brit. J. Urol.*, June, 1964, *36*, 211–219. (Address: Faculty of Medicine, Cairo University, Cairo, Egypt.)

Bilharzial affections of the ureter are discussed from a clinical, roentgenologic, and surgical standpoint, with emphasis on the pathogenesis and histopathology of the disease in the lower third of the ureter and on its surgical treatment. The author stresses the importance of proper diagnosis of the dilated atonic type of bilharzial ureter as contrasted to the more common obstructive or strictural bilharzial lesions mentioned in the literature. These atonic segments of the ureter require definitive surgical correction in the form of partial resection with end-to-end or end-to-side anastomosis rather than dilatation for a nonexistent ureteral stricture.

Three hundred and twelve cases were diagnosed, classified and treated. Pain in the form of renal colic together with frequency and dysuria represented the most common group of symptoms characteristic of obstruction and infection. Diagnosis was made by plain roentgenographic examination, intravenous pyelography, and cystoscopy. The findings of calcification in the wall of the ureter, in bilharzial papillomata, or within calculi were important diagnostically. Ureteral dilatation, stricture, and kinking were also noted on intravenous pyelography together with filling defects or papillomata, cysts or stones.

The case material is classified as follows: Ureteral stricture (77 cases); spindle-shaped ureter (54); dilated atonic tortuous ureter (48); calculus formation in a bilharzial ureter (35); trabeculated hypertrophic ureter (5); bilharzial peri-ureteritis (12); ureteritis cystica (12); bilharzial papillomata (13);

other conditions (56). A discussion of the histopathologic process in each of these conditions is presented.

Anti-bilharzial treatment was routine in all cases. However, all cases subsequently required surgical management. In stricture, operative dilatation with gum elastic or metal dilators is usually unsuccessful due to the tight nature of the bilharzial strictures. Various surgical procedures such as cystoscopic or transvesical meatotomy may be performed in strictures limited to the intramural part of the ureter. Definitive surgery with excision of the stricture and end-to-end or end-to-side anastomosis has the advantage of preserving normal ureterovesical function and preventing ascending infection. The author describes a new method of ureterovesical implantation which utilizes a mucosa to mucosa technique and minimizes the danger of regurgitation and ascending infection.—*Donald S Faust, M.D.*

VALVASSORI, GALDINO E., and PIERCE, ROBERT H. Osteosclerosis in chronic uremia. *Radiology*, March, 1964, *82*, 385-393. (Address: The University of Chicago, Department of Radiology, 950 East Fifty-Ninth Street, Chicago, Ill. 60637.)

A review of all cases with renal disease admitted to the University of Chicago Hospitals from 1948-1959 revealed osteosclerosis to be a relatively common feature in patients with chronic uremia. In this period, 987 patients were admitted with renal disease. Of these cases, there were 380 with an elevated blood urea nitrogen or nonprotein nitrogen but without associated disease which might alter the osseous findings. Roentgenograms on 280 of these were available for study. Of this group, in 219 cases a roentgenologically normal skeletal system was revealed. Abnormal changes were found in 61, decreased density of bones in 49 and osteosclerosis or mixed changes in 12. Bone changes were either reticular, granular or dense and mesh-like. In 2 of the 12 cases, there was evidence of renal rickets. In 4 cases, a roentgenologic follow-up of 3 years or more disclosed a progression from normal to various osseous changes.

The pathogenesis of the sclerotic changes in chronic renal disease is still obscure. Three pathogenic factors may be involved:

1. Anemia: A mild or severe form of anemia was noted in all cases of chronic renal disease.
2. Acidosis: In the early stages, demineralization occurs as the acidosis produces increased osteoclastic activity. As the disease progresses, a reciprocal response to the osteoclastic bone resorption may occur with stimulation of the osteoblasts.
3. Parathyroid: It is known that localized or diffuse osteosclerosis is occasionally observed in primary hyperparathyroidism side by side with the resorption of bone. Osteosclerosis has also been observed following prolonged administration of parathyroid extract to rats.

The roentgenologic and histologic findings suggest a gradual progression of the osseous changes from the osteoporotic or osteomalacic phase to pure osteosclerosis. The shortest time before the appearance of the osteosclerotic changes in this series was 3 years. With improved medical and surgical treatment of chronic renal insufficiency, more patients will survive long enough to develop osteosclerosis.—*A. W. Sommer, M.D.*

DENT, C. E., and FRIEDMAN, M. Hypercalcuric rickets associated with renal tubular damage. *Arch. Dis. Childhood*, June, 1964, *39*, 240-249. (From: Medical Unit, University College Hospital, London, England.)

Renal rickets or osteomalacia may be due to primary renal glomerular failure, to defects in renal tubular function or a combination of both. Both types are resistant to usual dosage of vitamin D, but the bone changes can be reversed following the administration of vitamin D in a dosage a thousand times the usual.

Rickets due to renal glomerular failure is associated with retention of phosphate and nitrogenous products. Calcium absorption from the intestinal tract is diminished. Therefore, fecal calcium content is high and urinary excretion of calcium is low.

Dent described 6 subgroups of tubular rickets. All were similar to each other in having a high renal clearance for phosphate. Glucosuria, aminoaciduria, inability to acidify the urine, defects in ammonia production, and renal loss of potassium were seen singly or in various combinations.

The authors present 2 cases of rickets which were not typical. Both showed increased excretion of calcium in the urine, whereas classic rickets cases have low urinary calcium. Malabsorption was excluded by the finding of normal fat excretion under balanced conditions. Both cases showed good absorption of calcium from the gut.

Hypercalcuria and rickets can occur in renal tubular acidosis, but this diagnosis was excluded by testing the patient's ability to handle ammonium chloride efficiently.

Both cases showed proteinuria, and electrophoresis demonstrated the protein to have the distinctive pattern seen in nonspecific renal tubular disorders.

The authors were unable to explain the etiology of the disorder which they assumed to be a limited defect of the renal tubules causing defective reabsorption of calcium, phosphorus, low molecular weight globulins and amino acids. Similar renal tubular damage has been seen in victims of heavy metal poisoning but no history or physical signs of such were found.

Both cases had reversal of bone changes after administration of moderate doses of vitamin D. However, growth retardation was evident in each patient. One of the two was mentally defective.

The authors have noted a high proportion of mental defectives in patients with congenital renal damage. Several of the previously described tubular defects are hereditary, but in these cases the family history was negative.—*Jay William Hammer, Jr., M.D.*

Kreuzer, H., and Sturm, A., Jr. Pseudopheochromocytoma. *German Med. Monthly*, May, 1964, *9*, 196–200. (From: First Medical Department, Medical Academy of Düsseldorf, Düsseldorf, Germany.)

Two cases of pseudopheochromocytoma are presented. The authors suggest that this entity be defined as "a disease displaying the symptoms of a pheochromocytoma with an increased production of catecholamines in the absence of a tumor of chromaffin cells."

In each case a young adult male hypertensive with symptoms suggesting pheochromocytoma was investigated. The first had bilateral flank tenderness, symptoms aggravated by bending over and an elevated urinary 3-methoxy-4-hydroxymandelic acid excretion with normal urinary catecholamines. Urinary studies were not done in the second case.

Presacral air insufflation with concurrent intravenous pyelography and laminagraphy demonstrated an abnormal shadow in the adrenal area above the left kidney in each patient.

At operative exploration, an aberrant mass of pancreatic tissue in the first patient and a tangerine-sized gastric diverticulum in the second patient were found adjacent to the normal appearing left adrenal gland and corrected. Postoperatively, the blood pressure of each patient returned to normal.

Pressure by the mass on the adjacent adrenal medulla with resultant increased secretion of catecholamines is suggested as the etiologic factor in the appearance of pseudopheochromocytoma.—*John T. Underberg, M.D.*

Skeletal System

Pillay, V. K. Ophthalmo-mandibulo-melic dysplasia: an hereditary syndrome. *J. Bone & Joint Surg.*, June, 1964, *46A*, 858–862. (From: General Hospital, University of Singapore, Singapore.)

The author describes 3 cases of ophthalmo-mandibulo-melic dysplasia, a hereditary anomaly thought to be transmitted as a mendelian dominant trait. The hereditary feature distinguishes this syndrome from arthrogryposis multiplex congenita and Möbius' disease.

Ophthalmo-mandibulo-melic dysplasia is characterized by: corneal opacities; temporomandibular fusion; absent mandibular coronoid processes; obtuse mandibular angles; and multiple bone changes in the limbs including radio-humeral and proximal radio-ulnar dislocation, and aplasia of the lateral humeral condyle, the head of the radius and the lower third of the ulna.—*John H. Harris, M.D.*

Wee, Lee Khee, and Pillay, V. K. Hereditary cleido—cranial dysostosis: with a note on the anomaly of cementum. *Singapore M. J.*, Mar., 1964, *5*, 3–9. (From: Departments of Dentistry and Orthopaedic Surgery, University of Singapore, Singapore.)

The authors report the occurrence of cleidocranial dysostosis in a Chinese family, affecting the father and 3 children in a family of 5 persons.

Four cases are presented with roentgenographic reproductions and descriptions of the major deformities in 3 of the cases.

All cases had defective and delayed dentition, absent outer ends of clavicles, and skull deformities. In the 2 sons there was also spina bifida occulta in the cervical dorsal spine and delayed ossification of the pubis, and 1 showed accessory metacarpal ossicles and long second metacarpals. The dental defects are described in detail and microphotographs of several of the teeth are presented which demonstrate the presence of acellular cementum.

The authors believe that this case complements the observations of Rushton who in 1956 suggested that a deficiency of cellular cementum was an additional dental defect characteristic of the dysostosis.—*James R. Knapp, M.D.*

Pollard, James J., and New, Paul F. J. Hereditary cutaneomandibular polyoncosis: a syndrome of myriad basal-cell nevi of the skin, mandibular cysts, and inconstant skeletal anomalies. *Radiology*, May, 1964, *82*, 840–849. (Address: Massachusetts General Hospital, Boston 14, Mass.)

The purpose of the authors is to describe, with emphasis on the roentgenographic findings, a rare hereditary syndrome consisting of myriad basal cell nevi of the skin, cysts of the jaw, and inconstant skeletal anomalies, and to add 6 new cases to the 20 reported in the English literature. They believe that it is important to alert the radiologists to the existence of this symptom-complex since it can be recognized in the presence of the proper combination of roentgenographic abnormalities. Because the syndrome bears no name, and a listing of the findings is lengthy and unwieldy, the authors propose the term "hereditary cutaneomanidbular polyoncosis."

The multiple basal cell nevi of the skin are slightly raised, flesh- to brown-colored, hard papules, usually 1 mm. to 1 cm. in diameter, and appear most commonly on the nose, upper eyelids, cheeks, trunk, arms, and neck, but may involve any part of the skin. Histologically, they are indistinguishable from basal cell carcinomata but they appear at a much earlier age than does the common basal cell carcinoma. They may be noted in early childhood but more commonly at puberty or in young adulthood. The cysts of the jaw are usually found in the mandible, most often in the ramus, but may occur in the maxilla. They are frequently multiple and almost always benign, but have a marked tendency to recur after apparently complete excision. These cysts are follicular or dentigerous and lined with epithelium that runs the gamut from a simple to a thickly keratinized stratified squamous cell type. They may appear in childhood or as late as the fifth decade but in general manifest themselves at an earlier age than do the skin lesions. The most common of the several skeletal anomalies are found in the ribs. Of these, the most frequent is broadening of the anterior end of a rib, with or without bifid configuration. A lamellar calcification or ossification of the falx cerebri occurs, which is quite distinctive in appearance as compared to the nonlaminated plaque type of ossification found in many normal skulls. These patients often present a characteristic facies with an increase in the distance between the inner canthi of the eyes. Often a mild degree of prognathism is seen.

The roentgenographic findings in the 6 cases presented by the authors are described. The cranial findings include: abnormal configuration in 2, dural plaques over the convexities in 3, bridged sella in 2, increased interorbital distance in 3, calcification of the falx in 4, and calcification in the tentorium in 3. In 1 patient there was bilateral shortening of the fourth metacarpals, a cervical rib and a dysplastic configuration of the medial third of a clavicle with loss of molding. Another patient had fusion of the vertebral bodies of some of the upper dorsal spine with vestigial and calcified intervening disks, reversal of normal upper dorsal kyphos, and spina bifida of D 1. Also present in this patient were rib anomalies and prominent medially projecting hooks of bone at the inferior angles of the scapulae. Another patient had only rib anomalies. In these latter 3 patients various cranial findings were also noted.

A brief history of this syndrome is given along with a good bibliography. Two photographs and several roentgenograms are reproduced illustrating many of the findings.—*Donald N. Dysart, M.D.*

SHULER, STANON E. Pycnodysostosis. *Arch. Dis. Childhood*, Dec., 1963, *38*, 620–625. (From: The Hospital for Sick Children, Great Ormond Street, London, England.)

Pycnodysostosis is a term coined by Marateaux and Laney to describe what is thought to be a variant of osteopetrosis.

The author presents 2 cases, 1 age 7½ years and 1 age 43 years, which are further examples of this syndrome. The adult is the maternal uncle of the child. The clinical findings are short stature, tendency of the bones to fracture easily, failure of the cranial sutures to close, short fingers and wrinkling of the skin of the hands. There also seems to be a genetic predisposition. Dental abnormalities of partial anodontia and faulty implantation may be other features.

The roentgenographic findings are increased density and enlargement of the cortex of the long bones; failure of the cranial sutures to fuse and large open fontanelles; sclerotic base of the skull; hypoplasia of the mandible and maxilla with loss of the angle of the mandible; hypoplasia of the distal phalanges and the acromial end of the clavicle; and increased density of the spine.

Differential diagnosis is limited primarily to osteopetrosis. Perhaps, Engelmann's disease, idiopathic hypercalcemia and vitamin D intoxication should also be mentioned, but none of these cause much problem in differential diagnosis.—*John L. Gwinn, M.D.*

PLASTINA, U., PANDOLFO, G., and CROSCA, A. Osteopatia da ipofosfatasemia: rilievi clinici e radiologici. (Osteopathy in hypophosphatemia: clinical and radiologic findings.) *Radiol. med.*, April, 1964, *50*, 341–350. (Address: Dott. Ubaldo Plastina, Via Marconi, Villa S. Giovanni (R.C.), Messina, Italy.)

The disease entity was first described by Rathbun in 1948 and called hypophosphatasia because of the serologic characteristics which distinguished it from all other bone diseases.

Two types, the early and the tardive, are described by various authors.

In the first, the infant appears normal at birth. After some weeks, anorexia, dyspepsia, weight loss and convulsions ensue. The skeletal roentgenologic examination discloses retardation of calcification of the cranial bones, wide areas of demineralization in the metaphyses and marked irregularity of the line of calcification. The epiphyses are osteoporotic and irregular in outline. The most interesting and most constant laboratory finding is the lowering of the serum alkaline phosphatase value which varies between 0 and 2 Bodansky units.

In the tardive type, the disease arises at the ages of 1 to 3 years or even later. The first manifestations are osseous and simulate rickets. The serum alkaline phosphatase is lowered to 1 or 2 Bodansky units and exceptionally lower than 1 unit.

The authors report a case of the tardive type.—*Anthony A. Blasi, M.D.*

PAUL, SHEILA SINGH, RAO, P. L., MILLICK, P., and KALLIANA, P. A case of chondrodystrophia calcificans congenita. *Arch. Dis.*

Childhood, Dec., 1963, *38*, 632–635. (From: Department of Paediatrics, Kalavati Saran Children's Hospital, Lady Hardinge Medical College and Hospital, New Delhi, India.)

Chondrodystrophia calcificans congenita (congenital stippled epiphyses, Conradi's disease or dysplasia epiphysialis punctata) is a rare but fascinating pediatric problem.

The authors describe a 6 week old infant who presented with a classic clinical picture of this disease. The more pertinent clinical findings included bilateral cataracts, shortened humeri and femora with flexion contractures of the joints. Roentgenograms demonstrated the characteristic stippling of the epiphyses in multiple areas of the body.

The article is recommended as a concise review of the clinical and roentgenographic findings of this disorder.—*John L. Gwinn, M.D.*

CHAWLA, S., and BERY, K. Iliac horns with arthrodysplasia and dystrophy of the nails—Fong's lesion: (review of the literature and report of a family). *J. Canad. A. Radiologists*, June, 1964, *15*, 60–64. (From: Lady Hardinge Medical College and Hospital, New Delhi, India.)

Six cases of this non-sex linked, dominant, hereditary trait are added to the 300 fully documented cases in the world literature. Five of these 6 cases showed evidence of pelvic dysplasia in the form of iliac horns. This term was originally coined by Fong in 1946, who described them as conical bony projections arising bilaterally from the posterior aspect of the iliac bones. The full syndrome comprises elbow, knee, and pelvic dysplasia along with dystrophy of the nails.

The authors point out that all components of this tetrad need not necessarily be seen in each individual case. They state, however, that iliac horns when seen are pathognomonic and call for a more extensive skeletal survey. Another finding that may be observed is hypoplasia or absence of the patella, with lateral displacement or permanent subluxation when present. Examination of the elbow shows hypoplasia of the capitulum, lateral epicondyle and radial head. Dislocation of the radial head may be present. Some patients may show hyperostosis of the frontal bones and a small sella turcica. Two of the cases presented in this article show such changes. Nail dystrophy is the commonest anomaly met in this syndrome and pelvic dysplasia is the least common manifestation encountered. The degree of pelvic dysplasia ranges anywhere from large iliac horns to the simple flaring of the iliac crests with prominent anterior superior iliac spines. All those who exhibit the classic findings are both physically and mentally normal and show no evidence of an impairment of their life span or a decrease in their fertility rates.—*Kenneth M. Nowicki M.D.*

JOYCE, JOHN J., III, and GRANT, DAVID B. Early diagnosis of slipping of the upper femoral epiphysis. *J.A.M.A.*, June 22, 1964, *188*, 1049–1052. (Address: Dr. Joyce, 5908 Greene Street, Philadelphia 44, Pa.)

The method of treating slipped upper femoral epiphyses is controversial but most authorities agree that results of late treatment are precarious. A review of the clinical findings in 26 cases revealed that trauma, degree and duration of slipping are important in determining the prognosis of the disorder. Limping and diminished hip motion were the most frequently encountered clinical defects.

Diagnostic delays resulted from misdiagnoses, family or patient reluctance to consult a physician, inadequate clinical examination, and roentgenographic misinterpretation.

Of the 26 cases reviewed, there were 14 boys and 12 girls. The age of onset varied from 10 to 16 years. Twenty-one of the 26 patients were obese. Endocrine imbalance is thought to be an important etiologic factor since children of the Fröhlich type or adolescents experiencing a spurt of rapid growth have been found to be quite susceptible.

Epiphysiolysis femoris must be excluded in any adolescent who limps or shows limitation of hip motion.—*Francis P. Shea, M.D.*

LICHTENSTEIN, LOUIS, and SAWYER, WAYNE R. Benign osteoblastoma: further observations and report of twenty additional cases. *J. Bone & Joint Surg.*, June, 1964, *46A*, 755–765. (From: Pathology Service of the Veterans Administration Hospital, San Francisco, and Department of Pathology, University of California School of Medicine, San Francisco, Calif.)

The authors present 20 cases of benign osteoblastoma which they have observed since a previous report by one of them (L.L.) on 11 cases. This total of 31 cases provides the material for an analysis of this bone tumor.

The majority of the cases was observed in children or teen-agers. Although the tumor was seen most frequently in the limb bones, the vertebral column, and the calvarium, it was also noted in other bones. In this group of 31 cases, tumors of the large limb bones and the vertebral column account for 60 per cent; those in the calvarium for 20 per cent; and those in the hand and foot bones for 20 per cent.

In a review of the roentgen features of this tumor, the authors take exception to other reports that this lesion has a characteristic roentgenographic appearance. A number of other lesions are mentioned which are not easily distinguished. It was observed that most of the lesions in these cases were well circumscribed and, even when expanding the bone, were bordered by a thin shell of bone.

The relationship of benign osteoblastoma to osteoid osteoma is briefly discussed and the two are believed to be distinct entities. There are 2 cases which developed eccentrically and were interpreted as being the periosteal counterpart of benign osteoblastoma.

Surgical treatment should be conservative. The treatment of benign osteoblastoma in the vertebral column, impinging on the spinal cord or its nerve roots, should be decompression followed by roentgen therapy.—*Donald E. Widman, M.D.*

Jewell, James H., and Bush, Leonard F. "Benign" giant-cell tumor of bone with a solitary pulmonary metastasis: a case report. *J. Bone & Joint Surg.*, June, 1964, *46A*, 848–852. (From: The Geisinger Medical Center, Danville, Pa.)

This case dramatizes a fact often reiterated that it is sometimes impossible to distinguish histologically between the benign and malignant giant cell tumor of bone.

A 30 year old patient showed roentgenographically an osteolytic expansile lesion of the distal radial metaphysis. The microscopic appearance of the curetted lesion was that of a benign giant cell tumor. Six months later on roentgenographic follow-up a soap-bubble recurrence was detected, and radiation therapy—3,000 r tumor dose—was given. One year later recalcification of the tumor site was observed.

Three years later a solitary lesion was discovered in his right lung. Segmental resection disclosed a metastatic giant cell tumor of low grade malignancy similar to that found in the radius, *i.e.*, "benign-appearing." Now 8 years after first admission the patient is doing well.—*Bernard Loitman, M.D.*

Dowling, Edmund A. Mesenchymal chondrosarcoma. *J. Bone & Joint Surg.*, June, 1964, *46A*, 747–754. (From: Department of Pathology, University of Alabama Medical College, Birmingham, Ala.)

The author reports 2 cases of mesenchymal chondrosarcoma. With this communication, a total of 13 cases of mesenchymal chondrosarcoma has appeared in the literature, the 2 described herein being unique by virtue of extraosseous origin.

Mesenchymal chondrosarcoma has been reported arising from ribs, flat bones, meninges and skeletal muscle in contrast to the usual chondrosarcoma that typically arises from the long bones of extremities. Clinically, a painful mass is the earliest manifestation. With increasing size, signs and symptoms referable to involvement or compression of adjacent structures become apparent. Local recurrence, especially following incomplete excision, occurs in approximately 40 per cent of cases. Distant metastases are usually general and may occur early or late (23 years). To date, the disease has been uniformly fatal.

The histologic appearance of the tumor is that of varying combinations of poorly differentiated mesenchymal cells and islands of recognizable chondroid tissue.—*John H. Harris, M.D.*

Phelan, John T., and Cabrera, Aurelio. Chondrosarcoma of bone. *Surg., Gynec. & Obst.*, July, 1964, *119*, 42–46. (From: Departments of Surgery and Pathology, Roswell Park Memorial Institute, and New York State Department of Health, Buffalo, N. Y.)

Twenty cases of chondrosarcoma of bone, each with at least a 5 year follow up or until death intervened, are reviewed.

The average patient's age was 46; males predominated 2:1. The two most common sites of occurrence were the pelvic girdle and thoracic cage; other sites were the long bones, clavicle, scapula and maxilla. The tumor presented as a firm, fixed, soft tissue mass, often painless, and occasionally fracturing pathologically. Size varied from 3 cm. to "massive." Growth was apparently slow and symptoms developed with larger size.

The roentgenographic appearance of 13 tumors was that of an area of bone destruction, cyst formation and a soft tissue mass with stippled calcification; 7 were described as "compatible with chondrosarcoma."

Grossly, the majority were cystic masses and a few were solid. Microscopically, 10 were composed of well differentiated cartilaginous tissue which could infrequently be mistaken for a benign enchondroma; 10 were poorly differentiated.

Nineteen tumors developed as primary lesions and 1 arose in a case of multiple hereditary osteochondroma. Ollier's disease and solitary exostosis or enchondromata have also been reported as predisposing lesions.

Surgery was the principal method of treatment. Twelve tumors were treated by local excision and each recurred locally. Seven of these were then treated by amputation or more radical local resection. Amputation was the primary treatment in 5 and irradiation in 2. Both irradiated tumors regressed but were not cured. Inoperable recurrent lesions and distant metastases were treated by irradiation with temporary regression, or with several chemotherapeutic agents without response. Pulmonary metastases occurred in one fourth of the cases and visceral metastases in 1 case. The local soft tissue infiltration of these tumors is emphasized.

Prognosis depends upon two factors: (1) the histologic grade and (2) the anatomic site of occurrence. Of patients with well differentiated neoplasms (10), 2 died and of those with undifferentiated lesions (10), 3 survived. Close proximity of the neoplasm to the axial skeleton limits adequate surgical treatment. Eleven patients survived 5 years and 9 are dead.—*John T. Underberg, M.D.*

BLACK, SAMUEL P. W., and KEATS, THEODORE E. Generalized osteosclerosis secondary to metastatic medulloblastoma of the cerebellum. *Radiology*, March, 1964, *82*, 395–400. (From: University of Missouri Medical Center, 807 Stadium Road, Columbia, Mo.)

Cerebellar medulloblastoma has a tendency to seed throughout the cerebro-spinal axis. Spread to sites outside the central nervous system, formerly considered very uncommon, has been reported however. With increasing frequency, metastases to bone are being observed.

The authors discuss the previously reported cases and present 3 additional cases. Their cases, aged 3 to 14 years, developed extensive osteosclerosis due to metastasis to bone following surgery and radiation therapy. In each instance, numerous bones were involved. The lungs, lymph nodes and viscera were normal.

It is hypothesized that the medulloblastoma is changed in some fashion by operation, radiation therapy, or other attendant environmental factors so that it acquires the ability to metastasize outside the central nervous system.—*A. W. Sommer, M.D.*

BELLER, HARRY E., and KIRSH, DAVID. Spondylolysis and spondylolisthesis following low back fusions. *South. M. J.*, July, 1964, *57*, 783–786. (From: Departments of Orthopedics and Radiology, University of Miami School of Medicine, Miami, Fla.)

Two cases of recurrent low back pain following lumbosacral fusion are reported. A 31 year old woman had spondylolisthesis at L3-4 following L4-S1 fusion, and a 36 year old man had spondylolysis at L3-4 following L5-S1 fusion.

The authors feel that this etiology of low back pain following fusion is often overlooked. With the lower lumbar spine fixed by bony union, there is undue downward and forward thrust upon the vertebra just above the fusion. This abnormal stress may produce spondylolysis without slipping, or slipping of the vertebral body without a bony defect. Repeated minor trauma is thought to be an important factor. Lateral flexion and extension roentgenograms as well as oblique views may be necessary to demonstrate the pathologic alteration.—*Mark D. Reiss, M.D.*

RHANGOS, WILLIAM C., and CHICK, ERNEST W. Mycotic infections of bone. *South. M. J.*, June, 1964, *57*, 664–674. (From: Departments of Orthopedics and Pathology, Duke University School of Medicine, and Veterans Administration Hospital, Durham, N. C.)

The authors anticipate an increase in the number of fungal infections of the bone as a result of antibiotic control of bacterial bone infections. They point out the similarity in the epidemiology of the more frequently occurring fungal infections in that they are acquired through soil contact, inhalation or entrance through the skin; also, that their clinical courses are protean and that the incidence of bone infection varies from 50 per cent in cases of blastomycosis to almost never in sporotrichosis. The diagnosis of these infections is chiefly by skin test, serologic test and by direct examination of biopsy or material from the lesions, all of which are more rapid and more accurate than culture methods.

Seven types of fungal infections are discussed as to clinical manifestations, roentgenologic features and treatment.

The roentgenologic manifestations are as follows:

1. *Actinomycosis.* Bone involvement is by direct extension from infected tissues. The lumpy jaw appearance which follows oral entrance is due to both periosteal and granulomatous reaction. The mandible shows little bony reconstruction and much destruction. In vertebral involvement, the body shows a "soap bubble" appearance with areas of lucency surrounded by normal or dense bone. Vertebral body collapse is rare and the disk spaces are maintained. In the case of burrowing sinuses reaching the bone, all parts of the vertebrae are involved. There is sometimes rib involvement and late periosteal reaction shows bridging between vertebrae.

2. *Blastomycosis.* Bone involvement runs 45–50 per cent with the vertebrae, ribs, skull, long bones, carpals and tarsals affected; in the subarticular areas the bone destruction is sharply defined from normal bone. Vertebral involvement is similar to tuberculosis with paravertebral abscesses and up to all parts of the vertebra involved. In actinomycosis the vertebral disk is often destroyed. There may be skip lesions due to prevertebral ligament dissemination.

3. *Coccidioidomycosis.* The bone is involved in 20 per cent of disseminated cases. All bones and joints except the temporo-mandibular joint, and the facial bones may be affected. Chief spots are at bony prominences at tendinous and ligamentous attachments. The roentgen findings are limited to the spongiosa. There are punched out lesions with sharply defined edges. There have been large confluent lytic areas reported. In spine involvement the disk is spared, and periosteal reaction is a late manifestation except in younger patients. Joints are involved by direct extension from the bone, with spaces in the weight bearing joints spared early in the infection.

4. *Cryptococcosis.* About 8 per cent of the cases show bone involvement. The vertebrae and cranium are the most often affected, but all bones may be involved. Both cortical and cancellous bone are destroyed with little new bone formation. The appearance is of a discrete area of destruction surrounded by a zone of dense bone. The rarely oc-

curring joint involvement is by direct extension.

5. *Histoplasmosis.* Bone involvement is extremely rare. In a generalized histoplasmosis, 40 per cent of the cases show bone marrow involvement without roentgenologic evidence.

6. *Sporotrichosis.* Bone lesions are rare. Two reported cases of knee joint involvement were very similar. They showed supracondylar femoral periosteal thickening, diffuse osteoporosis with punched out areas of radiolucency in the tibia and fibula. The joint space was decreased with loose bodies present.

7. *Mycetomas.* Destructive and regenerative bone changes are present due to the chronicity of the disease. The roentgenographic picture is reportedly typical. The second row of tarsal bones with the adjacent basilar portion of the metatarsals is involved most frequently. The shaft and head changes occur late. The early changes are loss of cortical definition with periosteal reaction manifested by spicule formation at right angles to reveal a "brush like" appearance. There is amorphous sclerosis of the spongiosa, with loss of the normal reticular pattern. Sequestra do not occur.

The authors also briefly discuss the treatment methods of each of these fungal infections of the bone.—*Sam W. Campbell, M.D.*

Blood and Lymph System

Lampe, William T., II. A severe reaction to angio-conray. *Radiology*, March, 1964, *82*, 456–458. (Address: York Hospital, York, Pa.)

Known toxic reactions to angio-conray which have been reported are reviewed. A case with death 48 hours after aortic injection of the medium is described, emphasizing that severe reactions do occur and that careful advance preparations for angiography is mandatory.

The case presented is that of a 58 year old white male with chief complaint of shortness of breath of 2 years duration, orthopnea, and chronic nonproductive cough. Past history revealed rheumatic fever at the age of 16 complicated by carditis and arthritis with one exacerbation at the age of 20. Positive physical findings were minimal cyanosis of the nail beds, clubbing of the fingers, limited thoracic expansion, increased anteroposterior chest diameter, and hyperresonant lungs. Examination of the heart revealed generalized cardiomegaly, palpable thrill over the aortic area, systolic and diastolic murmurs over the aorta along with a systolic murmur at the apex. Routine laboratory studies were unremarkable.

Percutaneous transfemoral retrograde aortography by the Seldinger technique was performed after sedation with phenobarbital, meperidine, and atropine. During insertion, the catheter inadvertently entered both the left subclavian and left common carotid artery with the catheter tip finally placed 1 cm. above the aortic valve. Test injection of 2 cc. of angio-conray was uneventful. Ten minutes later, 40 cc. of angio-conray was injected with the Cordis injector at 450 lb./sq. in. pressure. Immediately following injection, the patient became short of breath, pale, cold, and had difficulty speaking. Within 3 minutes paresis of the right side of face, right arm, and right leg was noted. The patient defecated involuntarily and lapsed into Cheyne-Stokes respiration followed within 30 minutes by complete left hemiplegia and coma. He died 48 hours later. Serum glutanic-oxalacetic transaminase was 165 units shortly before death, but postinjection electrocardiogram showed no change from admission tracings. Permission for necropsy was not granted.

The author states that no arteriosclerotic changes were noted in the aorta or the great vessels of the neck but admits that the inadvertent passage of the catheter into the left common carotid artery may have dislodged a plaque or caused vasospasm. However, he feels that there is no doubt that a serious immediate reaction to the contrast medium did occur, resulting in rapid deterioration and ultimate death.—*Howard R. Stewart, M.D.*

Meyer, Maurice W., and Read, Raymond C. Red cell aggregation from concentrated saline and angiographic media. *Radiology*, April, 1964, *82*, 630–635. (Address: Dr. Read, Wayne State University College of Medicine, 1401 Rivard Street, Detroit 7, Mich.)

As early as 1875, Zahn observed aggregation of red blood cells in the mesenteric circulation of the frog following topical application of salt crystals. In 1960, Raymond Read reported a similar phenomenon in dogs and cats following intravascular injection of concentrated salt solutions including angiographic media. This study was undertaken to provide further information in order to document previous preliminary microscopic observations.

Both *in vitro* and *in vivo* studies were made utilizing a glass capillary system, a plastic microchamber, and adult mongrel dogs. The mesenteric circulation of the dogs was microscopically examined and photographed both before and after intra-arterial injection of 2 to 3 cc. of a solution of either 15 per cent sodium chloride or 90 per cent hypaque. *In vitro*, a quantity of heparinized or oxalated whole blood (dog or human) or washed red cells suspended in saline was diluted with isotonic saline (control), 15 per cent saline, or 90 per cent hypaque by simultaneous injection into the plastic chamber. The mixing of the blood with the various test solutions was microscopically observed and photographed.

During the *in vivo* studies, the intravascular response was characterized by a series of practically simultaneous changes: (a) the velocity of blood flow decreased and small size aggregates of crenated red cells became visible in both the arterioles and small veins; (b) formation of large and small clumps produced hyperchromatism in the arterioles and temporarily plugged some of the small branches, occasionally producing pulsations in the arterioles and a change in the direction of flow, particularly in the small veins; (c) flow in the capillaries and venules became characterized by the conspicuous absence of the formed elements; small particles, however, possibly platelets, passed through these vessels; (d) central columns, often called "axial streaming," of red cells occurred in the small veins; (e) vascular dimensions remained essentially constant; however, during recovery some vasodilatation took place; (f) normal blood flow was usually re-established within 30 to 60 seconds.

In the glass capillary system a mixture of whole blood and isotonic saline showed no aggregation even at slow flow rates. With 15 per cent saline or 90 per cent hypaque, however, many large and small clumps interspersed with plasma were seen. With the microchamber, the point of mixing was observed revealing no aggregation with isotonic saline but both large and small aggregates with both test solutions.

In discussion, the authors recognize the work of others and present an adequate bibliography. They conclude that their present findings appear to be consistent with the hypothesis that the obstructive and ischemic effects of concentrated salt solutions and angiographic media are not related primarily to "vasospasm" but to red cell crenation and agglutination which result in increased internal friction of the red cell stream under conditions of increased viscosity.—*Howard R. Stewart, M.D.*

Whitesel, John A. Lymphography: its place in urology. *J. Urol.*, May, 1964, *91*, 613–618. (Address: 105 E. Centennial Avenue, Littleton, Colo.)

Lymphography, a diagnostic procedure of recent development in this country, must be brought into its proper perspective with a knowledge of its limitations and advantages in order that the physician may utilize the method more effectively. The major goals of lymphography are to detect the presence, localization, extent, and etiology of a disease process involving the lymphatic system. At the present time, the determination of the type of malignant tumor involving the retroperitoneal lymph nodes is more difficult than the determination of the extent of the involvement. Although metastatic carcinoma may be differentiated from lymphosarcoma, the pattern of the latter may also resemble that of metastatic seminoma. The high frequency of error encountered in the interpretation of the histologic nature limits its diagnostic accuracy in this respect.

The extent of the involvement is not always as accurate as would be desirable. Very small lymph nodes are sometimes not visualized and those lymph nodes which are completely involved by neoplasm are not demonstrated. In some instances where the radiopaque material has not been completely taken up by a lymph node, a mistaken diagnosis may follow and early filling defects may simulate normal variations.

One of the greatest contributions of lymphography has been the demonstration of the normal distribution of the retroperitoneal lymphatics, and as an aid to the surgeon in the removal of involved areas. Whereas, formerly unilateral retroperitoneal dissections were frequently done, there is today greater effort to do a bilateral retroperitoneal dissection for metastatic involvement from the urinary tract and particularly from primary testicular tumors. Evaluation of the surgical dissection can be monitored in the operating room by appropriate roentgenographic examinations.

Retroperitoneal lymphography has also been of considerable assistance in evaluation of radiation therapy for malignant metastasis in this region. Of lesser significance is the use of cytotoxic chemotherapeutic agents injected into the lymphatics, although further investigation of this procedure seems to be indicated.

The author gives a warning that a false sense of security may be produced by postoperative lymphograms, but that the procedure offers an additional method for the follow-up study of those patients with retroperitoneal neoplasm when treated by surgery, chemotherapy, or radiation therapy.—*George W. Chamberlin, M.D.*

Brody, H. Stephen, Helff, John R., and Bujdoso, Laszlo. Lymphangiography: an aid in urological surgery. *J. Urol.*, May, 1964, *91*, 606–607. (From: Urological and Radiological Services, Middlesex General Hospital, New Brunswick, N. J.)

The lymphatic vessels of each foot are cannulated with a #27 or #30 needle after localizing the vessels with the usual intracutaneous instillation of special dye. The authors then inject 20 cc. of ethiodol-chlorophyll slowly with the special injection apparatus. The injection is monitored in the leg by appropriate roentgenographic examination during the early minutes of the procedure. After 10 cc. has been injected, roentgenograms are made of the pelvis and chest. Subsequent roentgenograms are obtained at 2, 4, 8, and 24 hours. Oil embolism is a potential complication which is prone to occur in patients who have blocked lymphatics. Patients with poor pulmonary reserve should not have this study performed, as they may develop oil embolism of serious consequence. The purpose of the chlorophyll is to color the lymph nodes for surgical identification and removal.

The authors report the single case of a 35 year old man with a terato-carcinoma of the left testicle. The tumor was removed and a successful retroperitoneal lymph node resection was done by the aid of the green discoloration of the retroperitoneal lymph nodes and roentgenographic monitoring in the operating room during the procedure.—*George W. Chamberlin, M.D.*

Ortiz, Federico, Walzak, Myron P., and Marshall, Victor F. Chyluria: lymphatic-urinary fistula demonstrated by lymphangiography. *J. Urol.*, May, 1964, *91*, 608–612. (From: Department of Surgery (Urology), James Buchanan Brady Foundation of the New York Hospital, and the Department of Surgery (Urology) of Cornell University Medical College, New York, N. Y.)

Pyelolymphatic backflow is well recognized by radiologists when seen on the retrograde pyelograms and occasionally on the antegrade pyelograms. Chyluria is an uncommon finding and has been attributed in the past to a number of different causes. The recent development of retroperitoneal lymphangiography has facilitated the demonstration of the anastomosis with the renal lymphatics.

The authors present 2 case reports in detail and add a discussion of 8 additional patients with chyluria. All of the patients were born outside of the United States. Careful clinical and laboratory evaluation failed to demonstrate any evidence of filariasis. The chyluria was often quite variable and gross hematuria accompanied it in 4 of 5 cases. The influence of posture was studied in 5 patients in 3 of whom the urine was clear when the patient was maintained at bed rest. One of the patients was found to be shunting as much as 40 per cent of her dietary fat into the urine.

The article is illustrated by 6 composite figures and roentgenographic reproductions to show the anastomosis between the renal lymphatics, perirenal lymph nodes and the thoracic duct.—*George W. Chamberlin, M.D.*

Koehler, P. Ruben, Meyers, Wayne A., Skelley, Joseph F., and Shaffer, Burton. Body distribution of ethiodol following lymphangiography. *Radiology*, May, 1964, *82*, 866–871. (Address: Philadelphia General Hospital, 34th Street and Curie Avenue, Philadelphia 4, Pa.)

Lymphangiography using ethiodol has become a widespread procedure in recent years, but the distribution of the ethiodol in the body after leaving the lymphatic system is largely unknown, aside from its concentration in the lungs in the first 24 hours after injection.

The authors studied the problem of distribution of I^{131} tagged ethiodol in 15 dogs, sacrificed at intervals up to 17 days after injection. Various organs were analyzed for their I^{131} content, and an effort was made to determine the degradation of ethiodol.

The study showed a fairly straight curve of cumulative urinary excretion of I^{131}. The percentage of radioactivity in the arterial blood peaked at about 3 days, and then gradually declined. Free iodide was found in the circulation. The lungs apparently act as a filter, with about 50 per cent of the injected radioactive material found there, while 25 per cent was retained in the lymph system 3 days after injection. However, the ethiodol retained in the lymph system is released very slowly.

It was also concluded that if ethiodol with a high specific radioactivity were used for therapy, it would be very difficult if not impossible to achieve a uniform activity or to compute the dosage. In addition, there is a possibility of overirradiation of the lungs.—*J. C. Moore, M.D.*

GENERAL

McCort, James, and Vaudagna, James. Visceral injuries in battered children. *Radiology*, March, 1964, *82*, 424–428. (Address: Santa Clara County Hospital, San Jose, Calif.)

Several papers have appeared in the recent literature about the "Battered-child" or the "Unsuspected trauma syndrome." This term refers to an infant or child who has sustained physical abuse, usually repeatedly, from a parent or guardian. This paper is concerned with 10 infants or small children suffering initially unexplained visceral trauma. All patients presented an acute abdominal crisis with a vague and obscure or sometimes purposely misleading history being given by the parents.

The most common visceral injury, found in 7 of these 10 children, was rupture of the small bowel. Two sustained laceration of the liver, and 1 a perforation of the stomach. Laceration of the lung occurred in 1 child, and subpleural hemorrhage in another. Multiple viscera were traumatized in 5. With rupture of the intestinal tract, the roentgenographic findings were pneumoperitoneum, hemoperitoneum, and/or ileus. Considerable delay in seeking medical care in the 1 patient with a perforated duodenum resulted in the finding of multiple peritoneal abscesses. Hemoperitoneum was a striking finding in the 2 children with lacerated livers.

Also found in these children were various combinations of skeletal, craniocerebral, and soft-tissue injuries. Five had evidence of injury to the skeleton exclusive of the skull; in 2 there were numerous fractures. In evaluating the significance of multiple fractures, however, the radiologist must not forget that there are many diseases that will give a similar appearance. These include: vitamin C and D deficiency, leukemia, metastatic neuroblastoma, syphilis, osteogenesis imperfecta, hypophosphatemia, osteomyelitis, and cortical hyperostosis. Old or recent craniocerebral damage was found in 3 of the 10 children, and 5 were found to have multiple old and new bruises and contusions of various parts of the body. Three were found to be poorly developed, 1 was malnourished, and 2 had severe anemia, showing the presence of neglect. In 6 of the 10 cases, a study revealed a disturbance of the parent-child relationship.

The seriousness of visceral trauma in the battered-child syndrome is indicated by the fact that 4 of the 10 children in this study died. Since visceral trauma is most unusual in very young infants, an adequate explanation is necessary when this is found, especially if there is evidence of multiple injuries. If the battered-child syndrome is established, the hazardous situation in which these children live should be recognized and appropriate steps taken. The law requires the reporting of these findings to the law enforcement agency or the Juvenile Protection Service of the community. Removal of the child from his former surroundings may be necessary, or if this cannot be done, a program of continuous and regular supervision by the social worker will have to be undertaken.

The radiologist can be of great aid in the establishment of this syndrome by suspecting its presence and obtaining roentgenograms of the skull and long bones to detect evidence of repeated trauma and to provide a permanent legal record.—*Donald N. Dysart, M.D.*

GOIDANICH, I. F., and LENZI, LUCIANO. Morquio-Ullrich disease: a new mucopolysaccharidosis. *J. Bone & Joint Surg.*, June, 1964, *46A*, 734–746. (From: Departments of Orthopedics, the University of Pavia, Pavia, and the Istituto Ortopedico Rizzoli, Bologna, Italy.)

Morquio's disease is a congenital disorder of growth involving epiphyseal cartilage and bone derived from cartilage, resulting in dwarfism, kyphosis, genu valgum, and flattened vertebral bodies having tongue-like protrusions anteriorly. The epiphyses of long bones are small, irregular and fragmented. Other systems are not affected and intelligence is normal.

Hurler's disease, on the other hand, is a generalized connective tissue disease having similar but not identical roentgenographic changes as those seen in Morquio's disease. There is storage of mucopolysaccharides in many organs and clinically the patients have gargoyle facies, cloudiness of the corneas, deafness, hepatosplenomegaly, severe mental impairment, and skeletal changes producing dwarfism and kyphosis. One or more vertebral bodies in the thoracolumbar region are hooked.

Morquio-Ullrich disease indicates those cases presenting the external appearance and roentgenographic changes of Morquio's disease, together with one or more extraskeletal changes of Hurler's disease. There is also abnormal urinary excretion of the mucopolysaccharide, keratosulfate, which appears to be particularly important in the differential diagnosis since it is not present in cases of Morquio's or Hurler's disease.

Three cases of Morquio-Ullrich disease are presented with clinical and roentgen findings similar to those found in Morquio's disease and additional findings of cloudiness of corneas, hepatosplenomegaly, Reilly granulations in the leukocytes and urinary excretion of large amounts of keratosulfate.

The authors suggest that Morquio-Ullrich disease be classified as a mucopolysaccharidosis because of the clinical and biochemical findings. Further, they feel that the eponym, Morquio's disease, should be used only for those cases in which the locomotor system is involved and the urinary mucopolysaccharide pattern is not altered.—*Francis P. Shea, M.D.*

ZARNSTORFF, WILLIAM C., and ROWE, GEORGE G. Stereoscopic fluoroscopy and stereographic cineangiocardiography. *J.A.M.A.*, June 22, 1964, *188*, 1053–1056. (Address: Dr. Rowe, 1300 University Avenue, Madison, Wis., 53706.)

Utilizing a 9 inch image intensifier with square wave millisecond and fractional millisecond roentgenographic exposures, it has been possible to achieve stereoscopic perception. The right and left eye images are obtained by recording alternating images from a stereoscopic roentgen-ray tube containing focal spots approximately 2 inches apart. Direct stereoscopic viewing is obtained by using a cutaway circular mirror which, when rotated, alternately reflects and permits light to pass to a second mirror immediately behind. The rotation of the mirror is synchronized with the grid pulses of the 2 focal spots.

Cine recording of right and left eye images are obtained on alternate frames. Viewing of 35 mm. images was possible by polarizing alternate facets of the prism of a Tage-Arnoe projector and observing the projected image on a lenticular screen with polarized glasses.

Sixteen mm. viewing was made possible by using a rotating polarizing filter in place of the conventional shutter on a modified 16 mm. projector. An alternate and more acceptable method is modification of a Perceptoscope (Model 5102–2) to permit advancement and projection of 2 frames at a time. The images are polarized and converged by prisms. Viewing is through polarized glasses.

The method has proven of value in cinecardioangiography and reduces the necessity for multiplane examinations.—*Charles H. Helmen, M.D.*

BOHATIRCHUK, F. Use of x-rays in the detection of calcium in biological tissues at microlevel.

Canad. M. A. J., Dec. 7, 1963, *89*, 1171–1177. (From: Department of Anatomy, Faculty of Medicine, University of Ottawa, Ottawa, Ontario, Canada.)

The physical properties of x-ray in diagnosis at the macrolevel are also applied in x-ray morphology at the microlevel. Two methods of x-ray microscopy are most frequently used in medico-biologic research. These consist of x-ray projection microscopy, historadiography or contact microradiography with the resulting x-ray image studied microscopically.

Four items of equipment are necessary and are available: (1) a source of x-ray energy which generates x-ray in voltages from 8 to 12 kv.; (2) beryllium window tube; (3) fine grain emulsion films; and (4) a microtome for sectioning undecalcified bone, these sections should be no more than 10 μ thick.

The material presented consists of about 8,000 historadiographs and stained specimens from normal, pathologic and borderline conditions.

Calcium-poor areas are revealed by a blacker color than that of normal bone. This is especially significant when seen in serial sections. There does not appear to be any reliable histochemical reaction for the morphologic detection of calcium. In particular, small amounts of calcium, loosely distributed in tissue, can only rarely be seen in stained specimens.

In the author's opinion the data provided by historadiographs are more accurate in such cases and radiographs are probably the most specific available methods for the morphologic detection of calcium at the microlevel. The limited magnification of the x-ray image due to granularity of the emulsion is a noticeable defect. Also, no differentiation is possible between calcium carbonate and calcium phosphate.—*John L. Gwinn, M.D.*

RADIATION THERAPY

Walter, L., Harrison, C. V., Glucksmann, A., and Cherry, C. P. Assessment of response of cervical cancers to irradiation by routine histological methods. *Brit. M. J.*, June 27, 1964, *1*, 1673–1675. (From: Hammersmith Hospital, London; University of London; and Strangeways Research Laboratory, Cambridge, England.)

Valuable prognostic information can be obtained from ordinary routine examination of serial biopsies taken from patients undergoing radiation therapy for cancer of the uterine cervix. The authors devised a method of examining serial biopsies from tumors before and during radiation therapy in order to determine whether the early histologic changes induced by irradiation could serve as a means of assessing the radiocurability of the individual tumors.

A series of 210 patients with the diagnosis of squamous cell carcinoma, adenocarcinoma and anaplastic carcinoma involving the cervix was studied. The treatment planned involved the application of radium in 3 sessions alone or supplemented with external roentgen therapy. It was predicated on the stage of the disease, the type of tumor and the age and condition of the patient.

The patient had biopsies of the cervix done prior to treatment and at the time of subsequent radium placements.

The criteria evaluated to interpret prognosis were the presence of viable tumor cells, the presence of normal looking mitoses, and perceptible differences in degree and extent of differentiation between the pre- and post-radiation sections. An unfavorable response to treatment was indicated if the first post-radiation biopsy contained many viable tumor cells and especially if there were normal looking mitoses. The occurrence of a minor increase in differentiation was also evidence of poor response. A favorable response to treatment was indicated when there was an absence of normal looking mitoses in tumor cells and when viable tumor cells were scanty. An increase in the degree and extent of keratinization between the first and second post-radiation biopsies was also a favorable sign. Certain cases were recorded as doubtful since the post-radiation biopsies contained no recognizable tumor, but only necrotic debris or inflammatory exudate. They were recorded as doubtful also if there were tumor cells present, but the response was intermediate between good and bad as described.

The slides were evaluated by two independent groups, the Postgraduate Medical School and the Strangeways Research Laboratory. There was good agreement between the two groups and the recording of prognosis from both centers compared well with the 5 year survival rates.

The high correlation in prognosis based on quantitative analysis of radiation changes seen in serial biopsies of the cervix is not a laborious procedure and is within the scope of a routine pathology department. The striking differences in the clinical result for cases with an unfavorable and a favorable prognosis indicate that the assessment of radiation changes in serial biopsies can be of use to the therapist in deciding on additional treatment in individual patients.—*Luther W. Brady, M.D.*

Rimondi, C., and Mazzanti, G. Complicanze e postumi della radioterapia con alte energie dei tumori polmonari. (Complications and after effects of high energy radiation therapy of pulmonary neoplasms.) *Radiobiol., radioter. e fisica med.*, Nov.–Dec., 1963, *18*, 397–421. (From: Istituti di Radiologia e del Radio Università di Bologna, Bologna, Italy.)

In 207 cases of pulmonary neoplasms treated by a fairly uniform technique, the authors have sought to evaluate the complications which have arisen during or immediately after the completion of the treatment (cavitation of the neoplastic mass, radiation pneumonia, pleural reactive effusion etc.), and the late sequelae (pleuropulmonary fibrosis).

Generally, the cases studied were not amenable to surgical therapy.

In 18 per cent of the patients, treatment was suspended either because of worsening of the general condition or because of the development of distant metastases.

Eighty-eight patients who completed the treatment presented: (1) subjective improvement, *i.e.*, diminution or disappearance of dyspnea, of coughing and of pain; and (2) improvement of their general state, *i.e.*, gain in weight and a more normal blood picture.

Roentgenologically, one observed a reduction in size of the neoplastic mass, regression of the atelectasis and disappearance of the pleural effusion.

The early complications (pneumonia, cavitation and pleural effusion) were infrequent and responded favorably to adequate medical therapy.

Radiation fibrosis appeared after a period of a few months and was more severe in those cases in which the field was large (10 cm.×10 cm. or larger), or where the fractional daily dose was 500 r or greater.

One may consider fibrosis an after effect rather than a complication of radiation therapy; it represents substitution of dense fibrous tissue at the tumor site with a consequent functional impairment.—*Anthony A. Blasi, M.D.*

Reeves, Robert J., and Worde, Boyd T. Preliminary observations in cesium 137 teletherapy. *South, M. J.*, April, 1964, *57*, 379–385. (From: Department of Radiology, Duke University Medical Center, Durham, N. C.)

Cesium separated from U^{235} fission products consists almost entirely of stable Cs^{133}, beta emitter Cs^{135}, and beta and gamma emitters Cs^{134} and Cs^{137}.

The cesium 137 teletherapy unit used by the authors is a Caesatron. The tungsten diaphragm allows field sizes of 2×2 cm. to 10×10 cm. at 22 cm. source skin distance. A light source outlines the 55 per cent isodose level at 22 cm. source skin distance. The shutter mechanism is of the rotating wheel type. The source (CsCl) of 1,300 curies has a specific activity of 22 curie/gram, an active diameter of 2.89 cm. and an active heighth of 3.42 cm. At measurement, the exposure dose rate was 6.57 r/minute at 1 meter.

The major indication for Cs^{137} teletherapy with short source skin distance (22 cm.) is the lesion at depths of 5 cm. or less. High exposure dose rate, decreased bone absorption, skin-sparing effects, rapid decrease in percentage depth dose, decreased dose to deep underlying structures, and decreased integral dose are the advantages of this high energy radiation. Systemic reactions are rare, except for a "follicular" skin reaction, reflecting buildup of dose in a few millimeters of surface skin; other effects are the same as with Co^{60} teletherapy.

The procedure has been usefully employed in 21 postoperative carcinomas of the breast: 75 cases of bone metastases; 77 lymph node metastases and some other lesions. Satisfactory response in inguinal and axillary skin regions (ordinarily low tolerance areas) was observed. The doses ranged from 6,500 r to 7,500 r in 4 to 6 weeks.

Of 276 patients treated only 3 developed significant complications, all 3 receiving radiation for residual tumor in the oral cavity; 2 had radionecrosis of the mandible and 1 had subcutaneous fibrosis and necrosis.

The authors conclude that the basic principles in the indications and contraindications for the use of radiation therapy have not been altered by the use of beams of higher energies.—*Bernard Loitman, M.D.*

Duggan, H. E. Effect of x-ray therapy on patients with Peyronie's disease. *J. Urol.*, May, 1964, *91*, 572–575. (From: Department of Radiology, University of Alberta Hospital, Edmonton, Alberta, Canada.)

Plastic induration of the penis has been treated with irradiation for many years. This disease, which may be painful and deforming, seldom shows spontaneous improvement.

In a group of 87 patients, the author has applied roentgen therapy using a special technique to protect the gonads. The optimum dose of 1,000 r to each of 2 lateral portals over a period of 12 days is recommended. In 10 patients a second course of therapy consisted of 400 r to one lateral portal and 600 r to the opposite portal in a total of 5 days. The average time for any improvement after roentgen therapy was 6 months. In some instances, this period was as much as 18 months.

The long term results following a single course of roentgen therapy with this technique showed 83 per cent of the patients to be symptom free or improved. Of 10 who were given the second course of therapy, 2 were symptom free and 3 showed improvement.

Five tables are included in this article which analyzes the effects of radiation therapy on the various signs and symptoms of the disease.—*George W. Chamberlin, M.D.*

Duggan, H. E., and Weijer, D. L. Eustachian tube obstruction and radiation therapy. *Canad. M. A. J.*, June 27, 1964, *90*, 1454–1456. (Address: Dr. Duggan, Director, Department of Radiology, University of Alberta, Edmonton, Alberta, Canada.)

The results of external radiation therapy in the treatment of conductive deafness due to hyperplasia of lymphoid tissue obstructing the eustachian tubes are reported.

One hundred and ninety-four patients with eustachian tube obstruction were treated once a week for 4 weeks. The total dose to the skin for each lateral port was 600 r, and the midline dose for a skull of 12 cm. width was approximately 42 rad for every 100 r on the skin.

Of 121 patients under 15 years of age, 70 per cent were completely better or markedly improved at 6 months; 8 per cent showed no change. Long-term follow-up, averaging 4½ years after therapy, indicated that 82 per cent were completely better or markedly improved and 8 per cent were unchanged.

Of 73 patients over 15 years of age, 58 per cent were completely better or markedly improved at 6 months and 20 per cent showed no change. After long-term follow-up (4 years and 4 months after treatment) 68 per cent were completely better or markedly improved and 17 per cent showed no change.—*Bernard Loitman, M.D.*

PARKER, R. G., BURNETT, L. L., WOOTTON, P., and MCINTYRE, D. J. Radiation cataract in clinical therapeutic radiology. *Radiology*, May, 1964, *82*, 794–799. (Address: Section of Therapeutic Radiology, University of Washington School of Medicine, Seattle, Wash.)

Although there have been exhaustive animal studies, there is not a great deal of information about irradiation of the crystalline lens in the human eye with accurate knowledge of the dosimetry. The authors report their experience with 85 eyes of 43 patients who were treated for tumors of the head and neck, mostly with supervoltage apparatus. Treatment factors were extracted from records, and then the treatment situations were reproduced using a cadaver, with a small ionization chamber placed into the position of the lens. It was found that 4 out of 85 lenses showed changes that were felt to be due to ionizing radiation, and followed dosage of 2,000–3,900 rads delivered over a period of 46 to 85 days. Thirty lenses receiving over 500 rads in 32 to 93 days showed no changes, and 6 had equivocal changes at intervals from 2½ to 14 years following irradiation. Of 14 lenses receiving over 1,000 rads, 8 were without significant changes and 2 had equivocal changes at intervals of 5 to 13 years. Recognized changes in the crystalline lens of human eye incident to absorption of ionizing radiation are believed to be nonspecific. Other possible causes are often present, such as aging, systemic illness, pigmentary degeneration of the retina, and medication.

Adequate data are not available to establish thresholds for change in the lens of man due to ionizing radiation. Of clinical concern should be the incidence and severity of visual loss related to radiation change. The alternative to extensive irradiation of an eye is frequently orbital exenteration. If vision is satisfactory in the remaining eye, particularly in an elderly person, then the clinical result is acceptable.—*J. C. Moore, M.D.*

CAPSULAR ARTHROPLASTY for unreduced congenital dislocation of the hips

For your files...cut along this line

CAPSULAR ARTHROPLASTY—

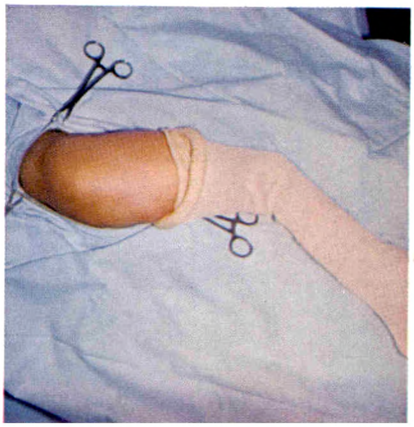
PROBLEM:

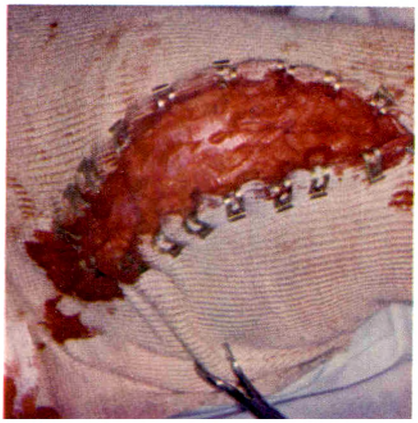
Unreduced congenital dislocation of the hip joints in a 4-year-old girl. Previous treatment by closed manipulation and open operation was unsuccessful.

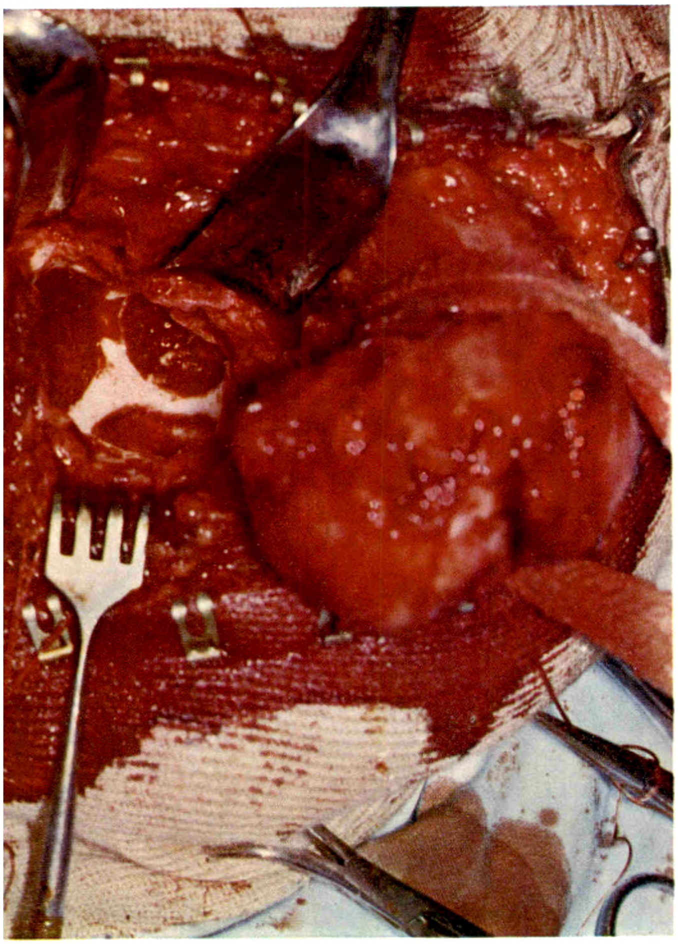
TREATMENT:

Bilateral capsular arthroplasties. The principle of preliminary traction established by Colonna was followed.

Figure 1. AP view of pelvis showing appearance of left hip 6 weeks after capsular arthroplasty has been performed. Right hip is *preoperative*. Steps in surgical reduction of right hip follow.

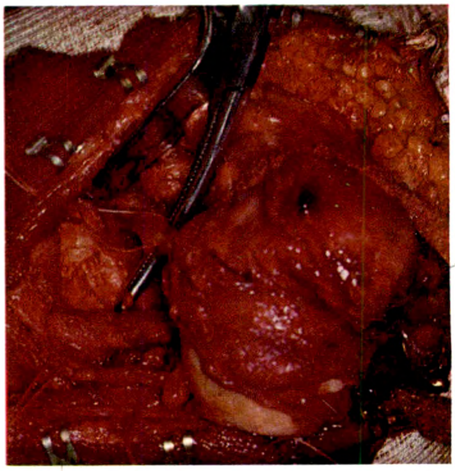






newly cut
diate cartilage.

Figure 8. Section of iliopsoas tendon marked by suture and clamp.



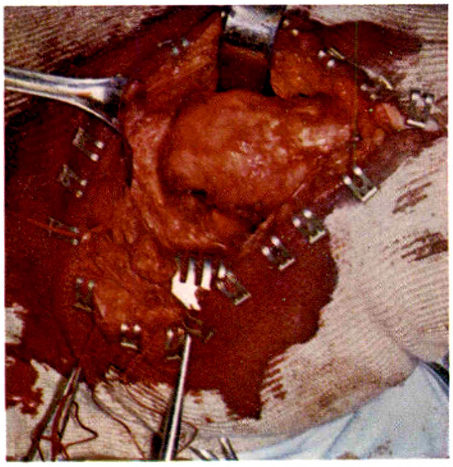


Figure 12. Reduction of capsule covered head into newly cut acetabulum.

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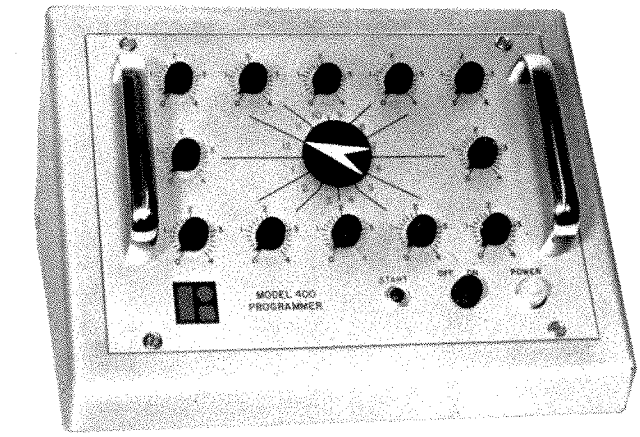

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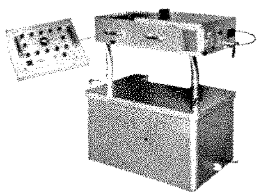

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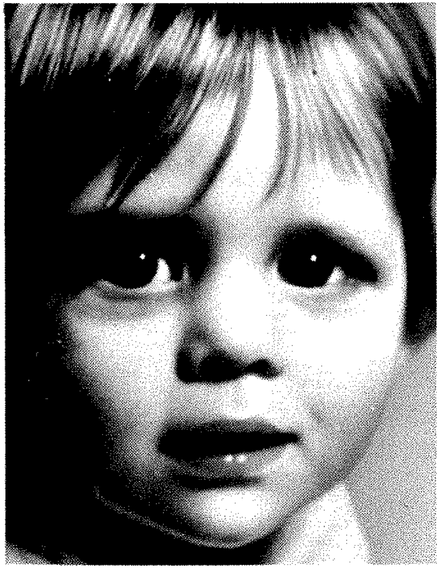

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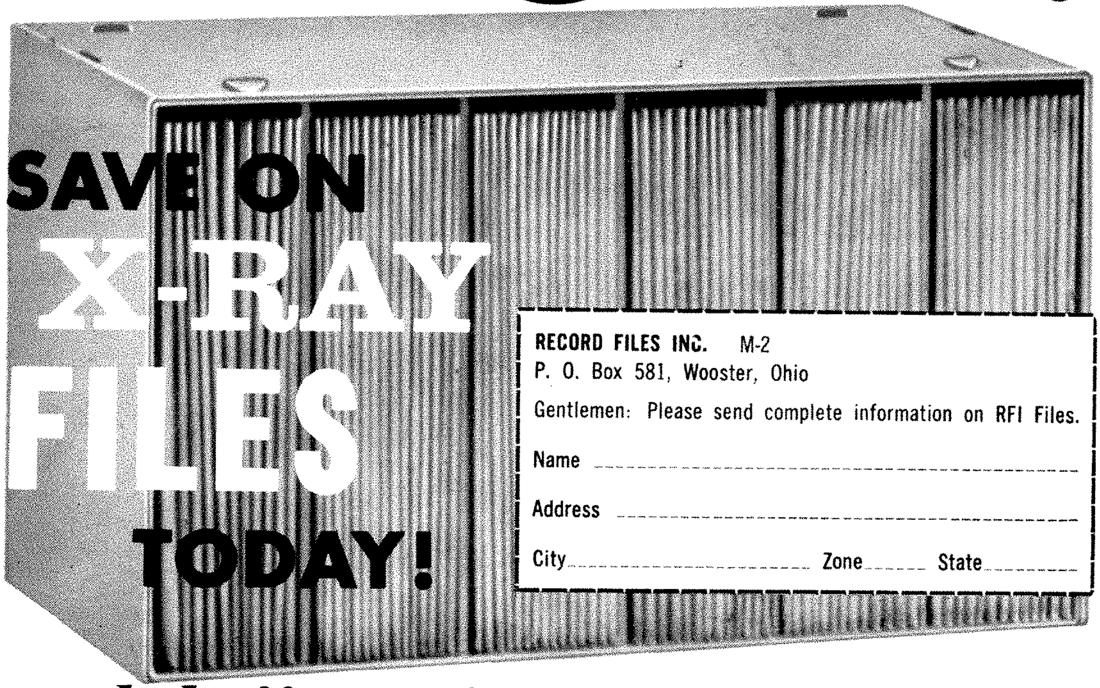

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Ansco (General Aniline & Film Corp.) ... vii
Atomic Energy of Canada Ltd. ... lxii
Automatic Seriograph Corp. (Division of Litton Industries) ... lv

Barnes-Hind Barium Products ... xxviii
Bell-Craig, Inc. ... xli

Cenco X-Ray Corporation ... viii, xxxii, xxxiii
Continental X-Ray Corporation ... xxxvi

Dunlee Corporation ... xiii

Eastman Kodak Company ... li, lii, liii, liv
Einstein, Albert, Medical Center ... lvii
E-Z-Em Company ... lxi

Fougera, E., & Company, Inc. ... xliv

General Electric Company ... ix, x, xi, xii, xxvi

Halsey X-Ray Products ... xx
Hanley Medical Equipment Company ... lvi

Ilford, Inc. ... xxv

Karger, S., AG. ... lviii
Keleket X-Ray Corporation ... xxxvii

Leedal, Inc. ... xxxix
Low X-Ray Corporation ... xix

Machlett Laboratories ... xlv
Mallinckrodt Chemical Works ... xxi, xxii, xxiii, xxiv
McElroy, Donald, Inc. ... lix
Mitchell X-Ray Company ... xl

North American Philips Company ... xvi, xvii
Nuclear-Chicago Corporation ... xiv, xv

Picker X-Ray Corporation ... xxix, xxxviii, Fourth Cover
Prince George Regional Hospital ... lvii
Profexray, Inc. ... i, 1

Record Files, Inc. ... lix
Reproduction Engineering Corporation ... xxx, xxxi

Schick X-Ray Company ... xlix
Siemens Medical of America, Inc. ... xliii
Standard X-Ray Company ... xviii
Squibb, E. R., & Sons ... v, xlii

Technical Measurement Corporation ... xlvii
Thieme, Georg Verlag ... xlvi
Thomas, Charles C, Publisher ... xlviii, lvi

U. S. Radium Corporation ... xxvii

Varian Associates ... xxxiv, xxxv

Walker Industries ... vi
Westinghouse Electric Corporation ... Second Cover
Winthrop Laboratories ... Third Cover

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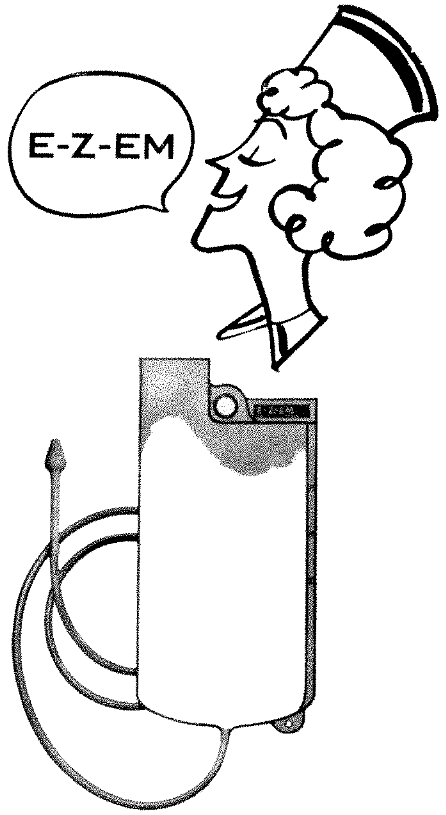

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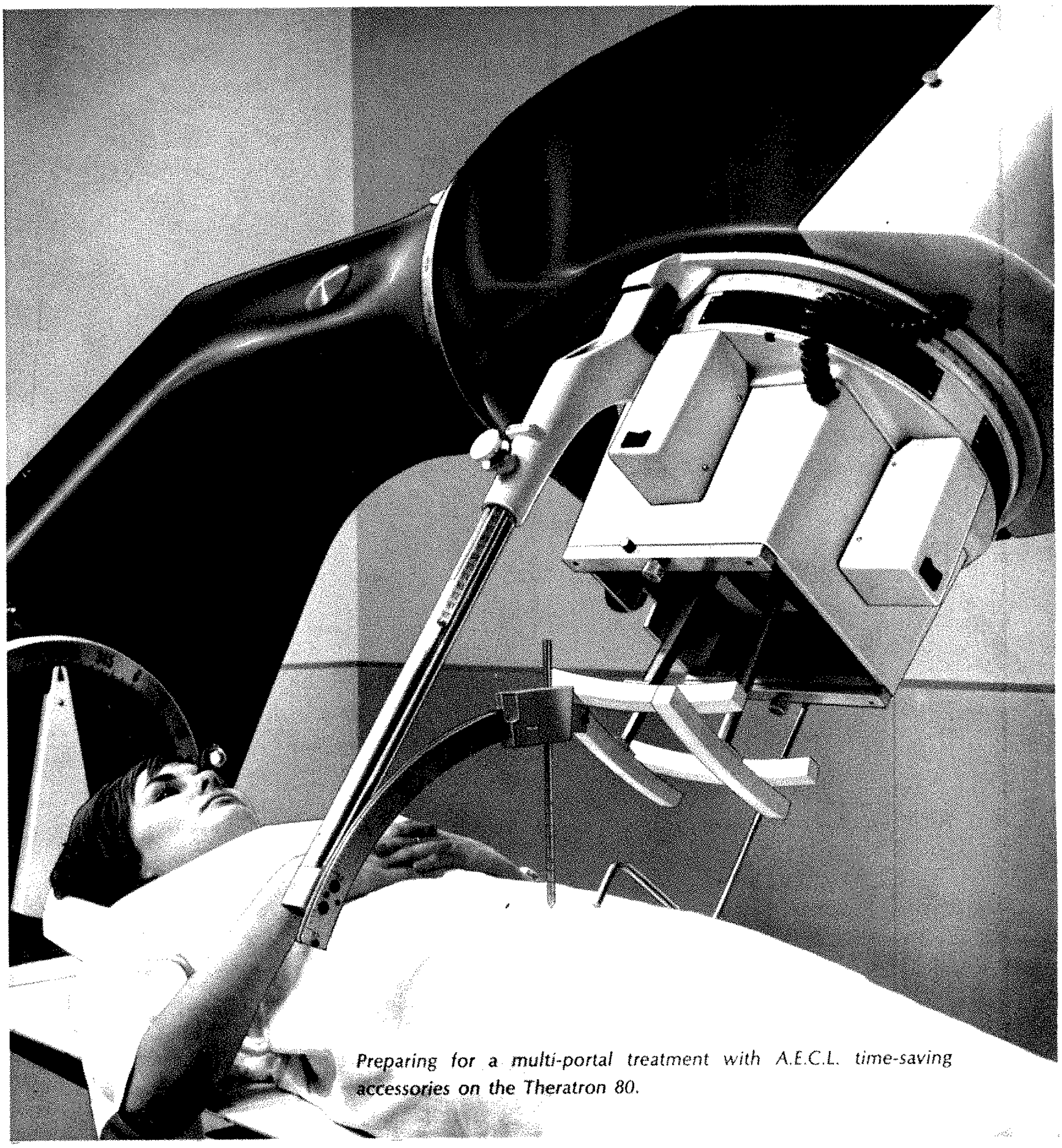

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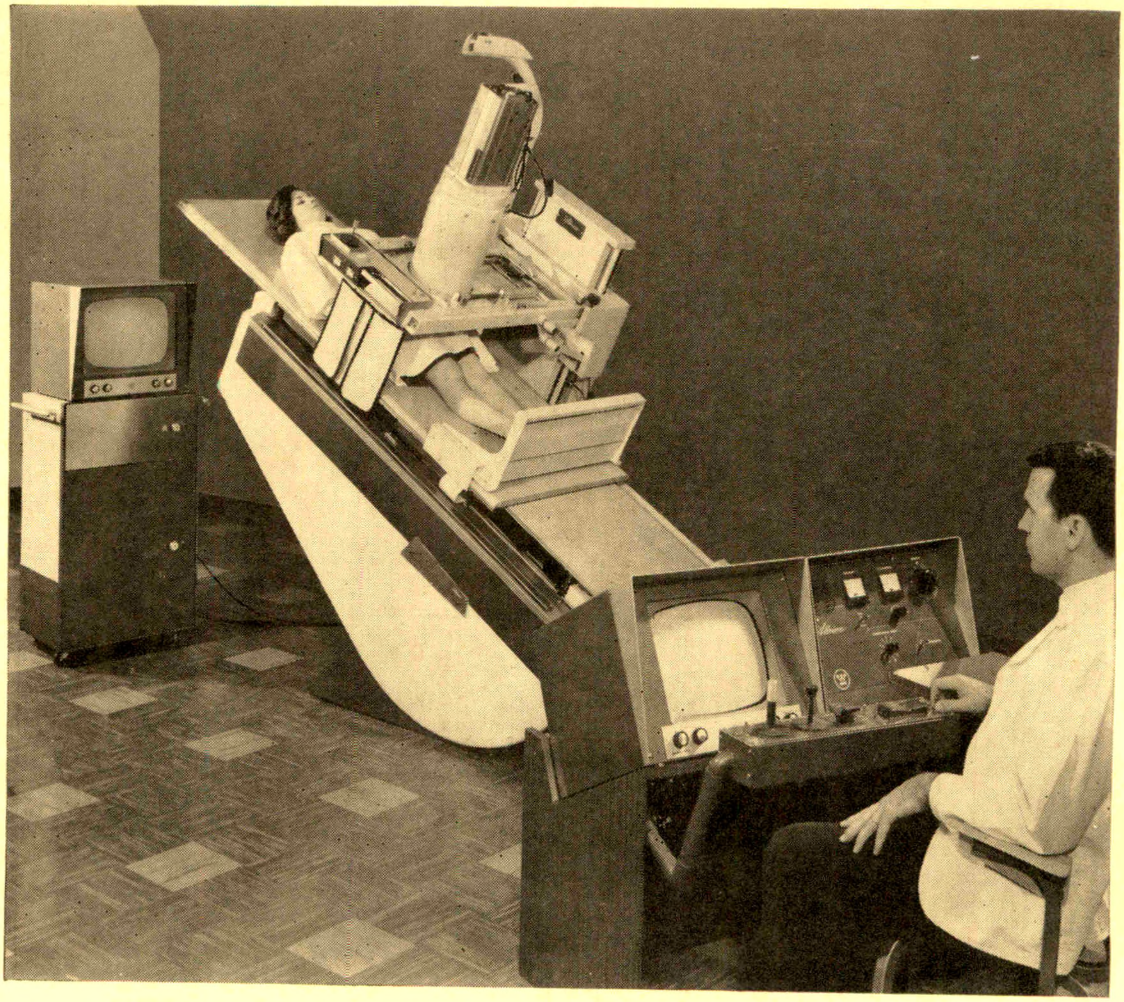

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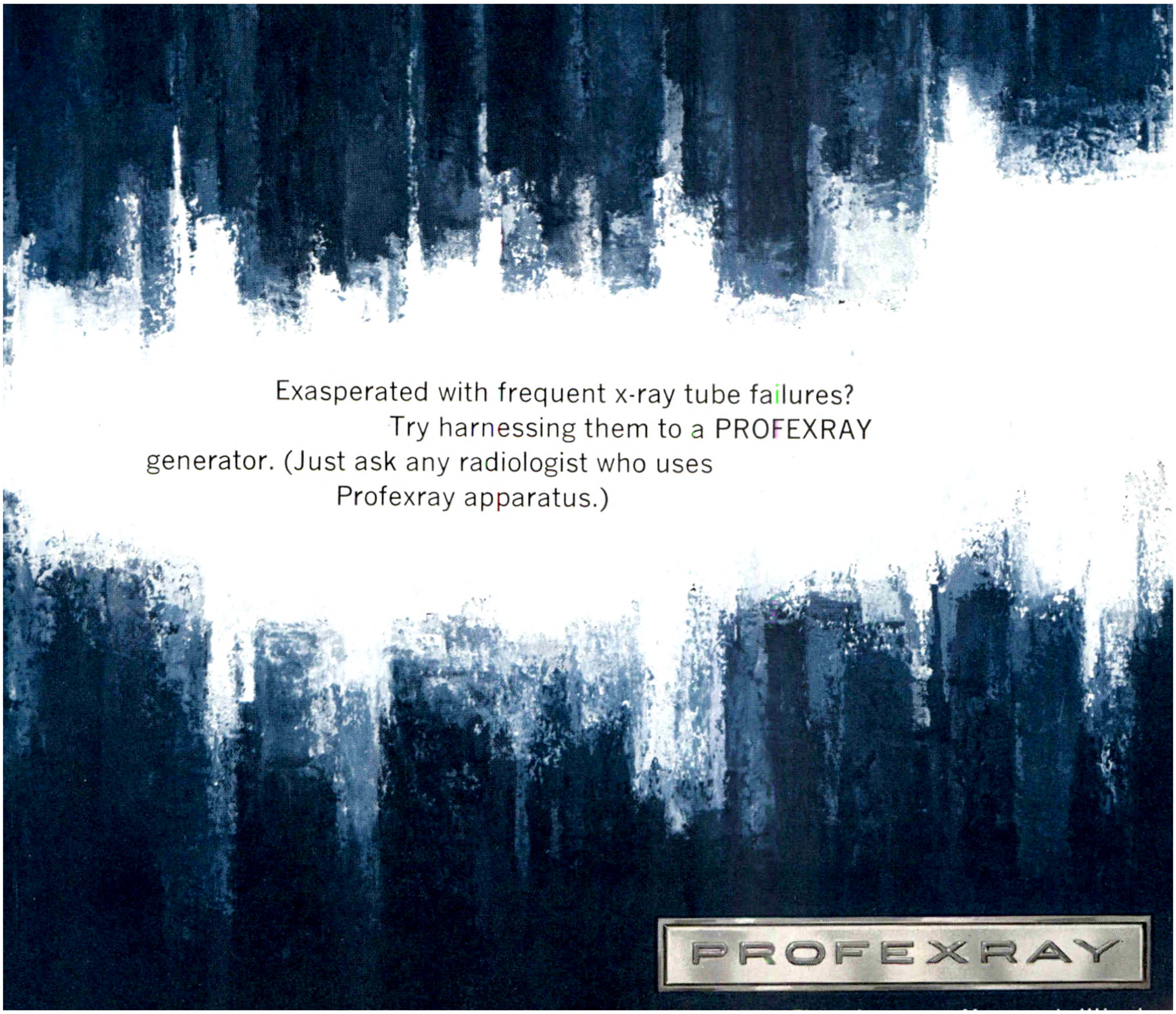

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CONTENTS

Recent Cinefluorographic Advances in Palatopharyngeal Roentgenography. ROBERT F. SLOAN, B.A., STANLEY W. BRUMMETT, M.D., JOSEPH L. WESTOVER, M.D., ROBERT M. RICKETTS, D.D.S., M.S., AND FRANKLIN L. ASHLEY, M.D. 977

Combined Pressure Tracings and Cineroentgenography of Hollow Muscular Viscera. LUIGI TURANO, M.D. 986

Esophageal Reflux. GERALD M. VANDERVELDE, M.D., AND HARLEY C. CARLSON, M.D.. 989

Congenital Esophageal Strictures. WILLIAM G. GREENOUGH, M.D.. . . . 994

Esophageal Varices in Superior Vena Caval Obstruction. DONALD L. OTTO, M.D., AND RAYMOND S. KURTZMAN, M.D. 1000

Herniation of the Transverse Colon through the Foramen of Morgagni Simulating Left Ventricular Hypertrophy; Report of a Case. DAVID C. HILLMAN, M.D., MAURICE M. REEDER, CAPTAIN, MC, AND OSCAR S. DEPRIEST, III, MAJOR, MC 1013

Transmigration or Prolapse of Esophageal Mucosa. WALTER J. RUSSELL, M.D., AND TAKASHI KOGURE, M.D. 1017

Recent Advances in the Removal of Magnetic Foreign Bodies from the Esophagus, Stomach and Duodenum with Controllable Permanent Magnets. F. E. LUBORSKY, B. J. DRUMMOND, AND ARTHUR Q. PENTA, M.D. . 1021

Glomus Tumor of the Stomach; A Case Report. HAROLD J. SCHNEIDER, M.D. 1026

Retroperitoneal and Mediastinal Chemodectoma; Report of a Case and Review of the Literature. SEYMOUR HABER, M.D. 1029

The Intestinal Knot Syndrome. LUCEIL B. NORTH, M.D., AND H. STEPHEN WEENS, M.D. 1042

Small Intestinal Diverticulosis. LAWRENCE H. CAPLAN, M.D., AND HAROLD G. JACOBSON, M.D. 1048

Intramural Intestinal Hemorrhage. M. T. KHILNANI, M.D., R. H. MARSHAK, M.D., J. ELIASOPH, M.D., AND B. S. WOLF, M.D. 1061

Unusual Small Bowel Diseases; Methods and Observations. GILBERT S. STACY, M.D., AND JOHN W. LOOP, M.D. 1072

(*Continued on page iv*)

Experimental Infarction of Small Bowel in Dogs; Roentgenographic Demonstration with Water-Soluble Iodine Contrast Medium. Bruce Vest, M.D., and Alexander R. Margulis, M.D. 1080

The Meandering Mesenteric Artery of the Colon. Myron Moskowitz, M.D., Harold Zimmerman, M.D., and Benjamin Felson, M.D. 1088

Indications for Selective Celiac and Superior Mesenteric Angiography; Experience with 128 Cases. R. A. Nebesar, M.D., J. J. Pollard, M.D., L. H. Edmunds, Jr., M.D., and C. F. McKhann, M.D. . . . 1100

Gastrointestinal Manifestations of Abdominal Aortic Aneurysms. Fred K. Sondheimer, M.D., and Israel Steinberg, M.D. 1110

Electrolyte Depletion Syndrome Associated with Villous Adenomas of the Rectosigmoid. Lawrence R. Cooperman, M.D., and Robert M. Lowman, M.D. 1123

The Inclined Lateral Decubitus Position for Cholecystography. J. R. Amberg, M.D., F. F. Zboralske, M.D., and E. R. Johnson, R.T. . 1128

Percutaneous Transhepatic Cholangiography; A Preliminary Report Describing a New Approach to the Examination. Ernest J. Ferris, M.D., Julio Joison, M.D., Jerome H. Shapiro, M.D., and John J. Byrne, M.D. 1131

Canine Cineportography. Philip M. Johnson, M.D., Richard L. Murtland, M.D., and Kenneth Weaver, M.D. 1139

Hamartoma of the Spleen; Angiographic Observations. Lewis Wexler, M.D., and Herbert L. Abrams, M.D. 1150

Radioisotope Photoscanning in the Diagnosis of Pancreatic Disease. Gerald Burke, M.D., and M. S. Goldstein, M.D. 1156

Portal Vein Gas: A Grave Prognostic Sign. Robert G. McCandless, M.D. 1162

Progressive Postoperative Pneumoperitoneum; Air Entering through Drain Sites. D. James Heslin, M.D., and Ronald A. Malt, M.D. . . 1166

Nonfatal Intravasation of Barium. Herbert R. Zatzkin, M.D., and Gerald A. L. Irwin, M.D. 1169

Microangiographic Study of Gastrointestinal Lesions. Harlan J. Spjut, M.D., Alexander R. Margulis, M.D., and William H. McAlister, M.D. 1173

Physical Factors in Cinefluorography; Exposure Meter and Phantom Materials. John Hale, Ph.D., and David L. George, B.S. 1188

Physical Factors in Cinefluorography; Relative Patient Dose for 35 mm. and 16 mm. Camera Systems. John Hale, Ph.D., Robert D. Epperson, A.B., David L. George, B.S., and Theodore A. Tristan, M.D. . 1192

Officers 1199

Editorial

Barium or Air for Examination of Colon. Seymour Fiske Ochsner, M.D. 1200

News Items 1202

Book Reviews 1203

Books Received 1204

Society Proceedings 1206

Abstracts of Radiological Literature 1211

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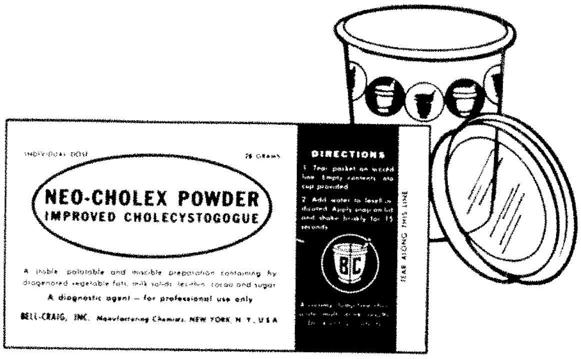
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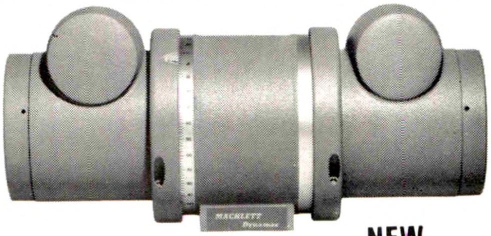

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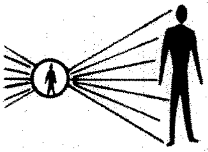

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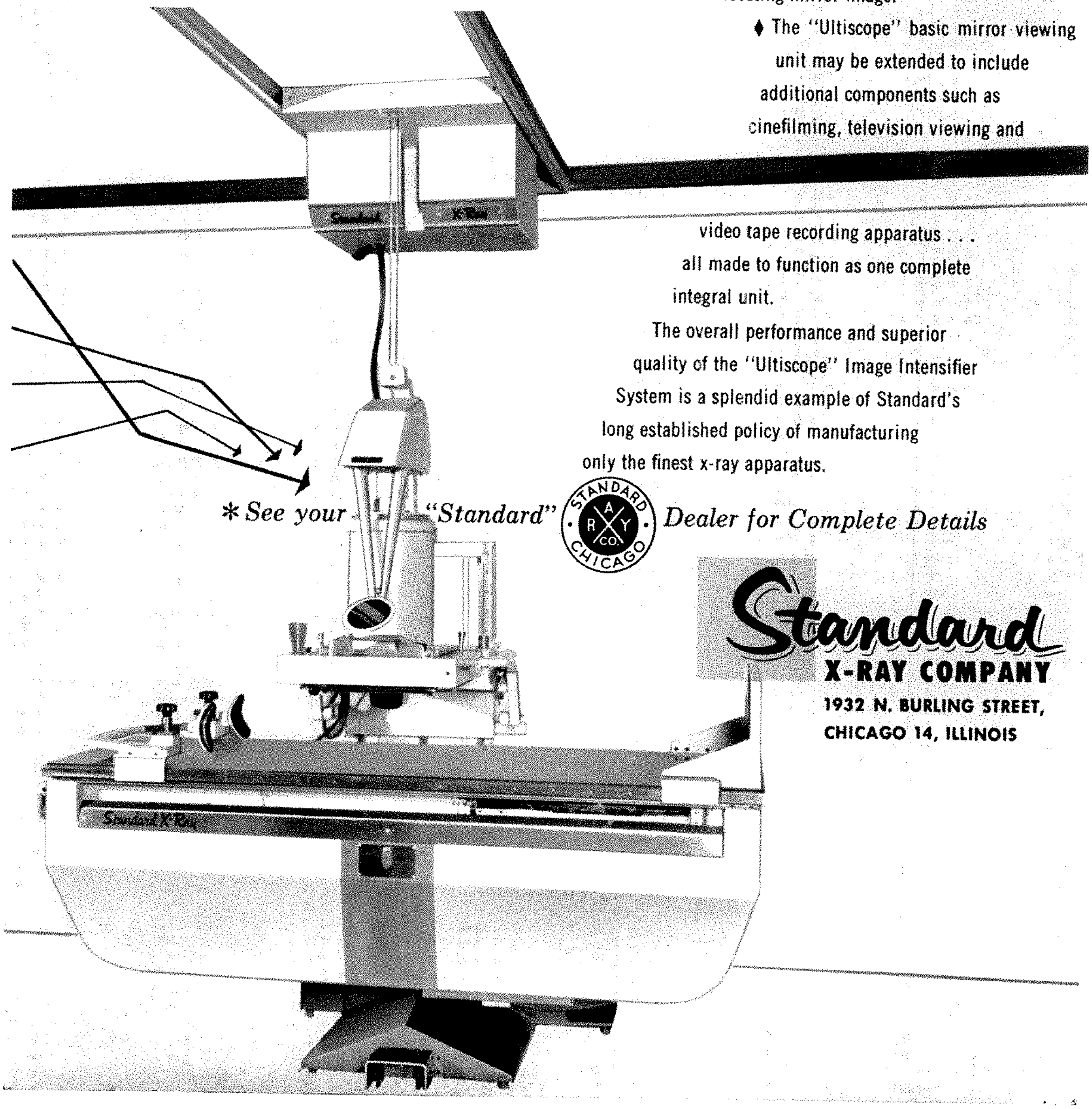

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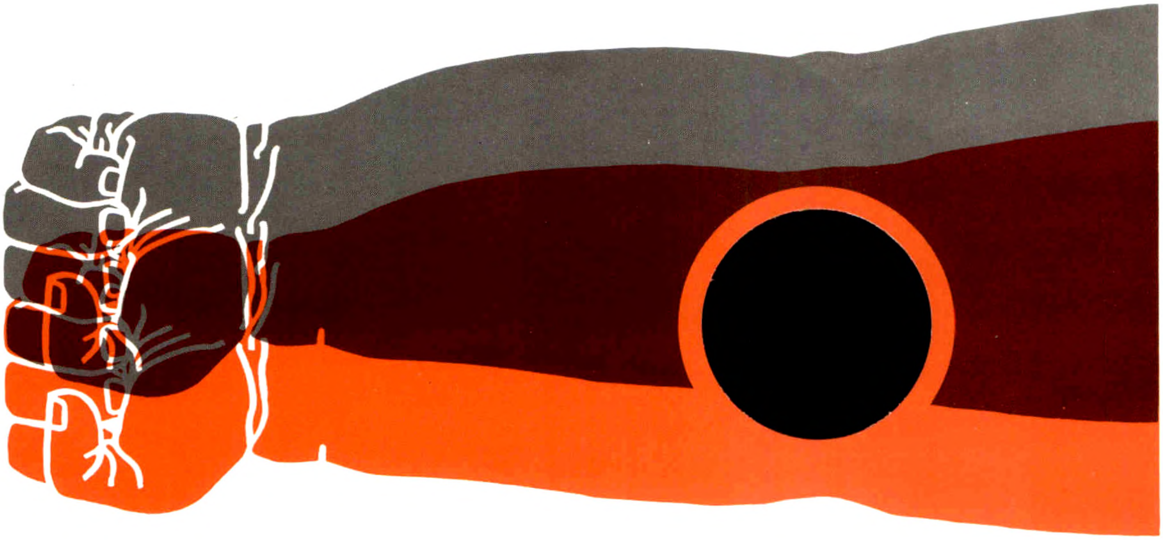

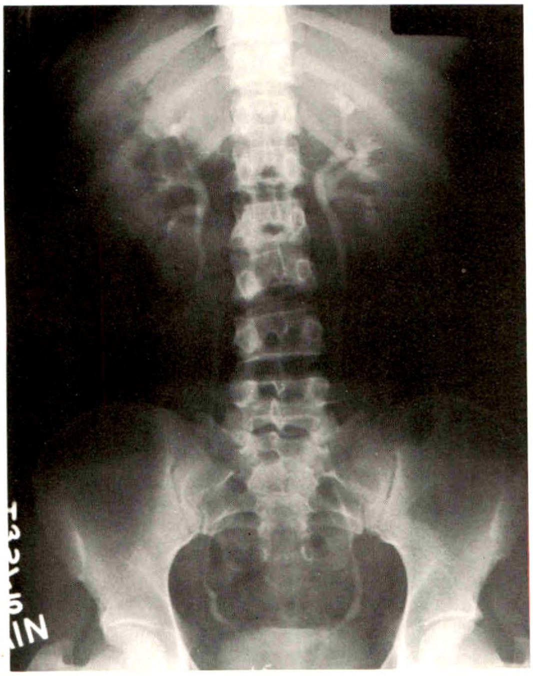

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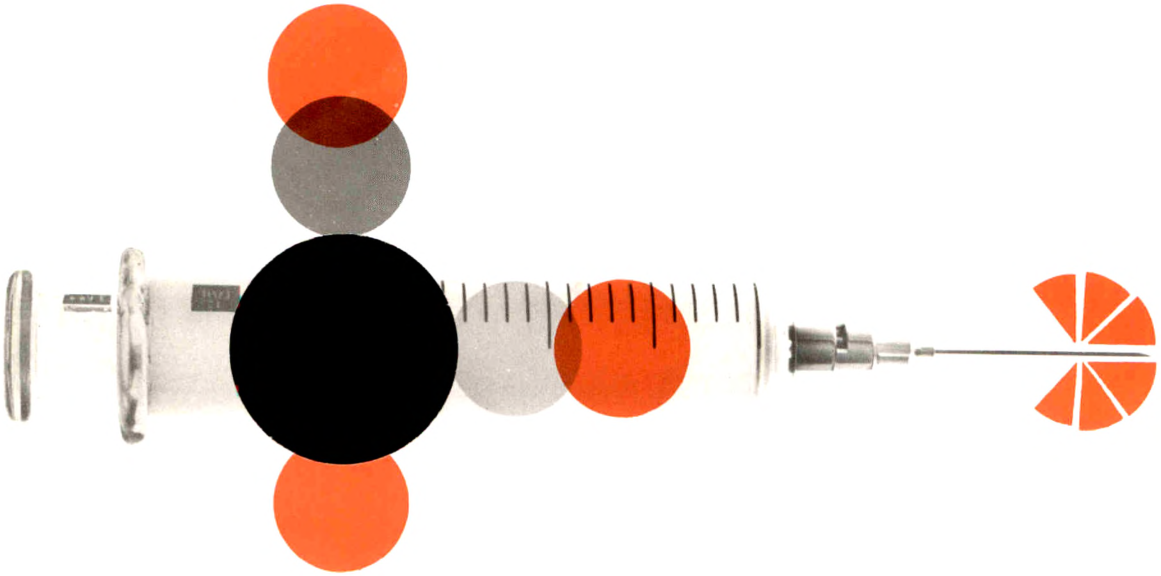

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Caution: CONRAY-400 (sodium iothalamate 66.8%) and Angio-CONRAY (sodium iothalamate 80%) are not intended for use in cerebral angiography. For this purpose, CONRAY (meglumine iothalamate 60%) is recommended.

Contraindications: For all three media, caution is required in cases involving severe systemic disease. In patients with advanced renal disease, use with caution and then only when need for examination dictates. In patients with a history of allergy or those exhibiting a positive reaction to the sensitivity test, drugs and facilities for emergency use must be readily available.

Precautions: For Angio-CONRAY (sodium iothalamate 80%) and CONRAY-400 (sodium iothalamate 66.8%), cardiac arrhythmias, including ventricular fibrillation, have been reported with the use of these media in procedures for visualizing the heart and its associated large vessels. Such occurrences may be associated with situations in which appreciable amounts of relatively undiluted medium enter the coronary circulation. Facilities for immediate treatment of such reactions should be available.

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RECENT CINEFLUOROGRAPHIC ADVANCES IN PALATOPHARYNGEAL ROENTGENOGRAPHY[*]

By ROBERT F. SLOAN, B.A., STANLEY W. BRUMMETT, M.D., JOSEPH L. WESTOVER, M.D., ROBERT M. RICKETTS, D.D.S., M.S., *and* FRANKLIN L. ASHLEY, M.D.
LOS ANGELES, CALIFORNIA

SINCE the development of practical roentgen-ray image intensifiers in 1953, cinefluorography has become an eminently practical method of roentgen diagnosis.[21] More than 800 published reports can now be found in the scientific literature which describe various uses of roentgen-ray cinematography.[33] Cinefluorography, complemented by spot roentgenographic techniques and cephalometric films, is a practical and important means of pharyngeal assessment because it provides three basic data: (1) motion of the oral and pharyngeal structures; (2) variations in their roentgenographic density; and (3) a chronology of the changes occurring during a functional cycle.[25]

In this paper (1) a brief description of the more recent advances in cinefluorographic filming techniques for studying the physiology of the cranial and pharyngeal areas, including palatal incompetencies and postlaryngectomized patients, and (2) the basic cinefluorographic diagnostic techniques used in the study of the pharyngeal regions are presented.

RECENT ADVANCES IN CINEFLUOROGRAPHIC TECHNIQUES

The references to the trends and technical advances in cinefluorography have been numerous.[3,6,7,15,30] Roentgenography and cinefluorography as aids to diagnosis of palatal incompetencies and other pharyngeal problems are not new. Extensive clinical studies are available in the radiologic literature on the subjects of deglutition[1] and phonation.[1,8,9,18–20] These publications present numerous case reviews of still roentgenographic studies of speech problems, including (1) palatal incompetencies (cleft palates, postbulbar defects, palsy, poliomyelitis, palatal paresis, pharyngeal stenosis), (2) hypernasality (submucous cleft palates, postoperative complications from tonsilectomies and adenoidectomies),

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[*] From the School of Dentistry, the Division of Plastic Surgery, and the Department of Radiology, University of California, Los Angeles, California, and Saint John's Hospital Research Foundation, Santa Monica, California.

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and (3) other growth and development anomalies of the nasopharyngeal regions that have caused speech defects.

Roentgenologic observations, despite many limitations, have provided the most objective evidence for the evaluation of the accepted speech rehabilitation procedures used for postlaryngectomized patients. The majority of roentgenographic studies on the laryngeal region have been restricted to roentgenography.[2,4,10,12,14,17,28,29,32] Only a limited number of cinefluorographic studies have been reported;[5,11,16,24,27] to date, comprehensive studies documenting standard cinefluorographic procedures have not been reported. Damste, quoted by Levin,[14] compared roentgenograms of 8 good postlaryngectomized speakers with those of 8 poor speakers, and noted that the shape of the pseudoglottis is fully determined by the cricopharyngeus muscle. In most cases, the prominence formed by this muscle is prior to the fifth and sixth vertebrae; however, in poor speakers it is often lower. He also observed that an important characteristic of the poor speaker is the formation of a bulge in the ventral side of the pseudoglottis in which secretions easily accumulate. As a result, the voice produces a bubbly mucoid sound.

Robe *et al.*[22] used roentgenography to study the pseudoglottis in 16 laryngectomized patients. Five roentgenograms were made of each subject. The following observations were made from the series of roentgenograms obtained: (1) the films of the good speakers showed a constriction of the pharyngoesophageal air column at the approximate level of the fifth vertebra and the cricopharyngeus muscle; and (2) the films of the good speakers also showed an air column of adequate size below the pseudoglottis. The formation of a column of air below the pseudoglottis appeared to be analogous to the use of subglottal air in normal phonation. Such columns of air were absent on the films of the poor speakers. An air column of adequate size is important in the production of a good esophageal voice.

In 1955, cineroentgenograms of postlaryngectomized patients were produced by Van den Berg, Moolenaar-Bijl, and Damste. However, after a review of the world literature, only a few other such cineroentgenographic studies were found.[2,4,5,11,24,31] Hodson and Oswald[11] observed that during examination the sphincter at the upper end of the esophagus was in a constant state of activity, varying rapidly in tightness with the modulations of speech. They also noted that, although the length of various sphincters differed only slightly from case to case, the position of the center of the lumen varied considerably. A possible explanation offered for the varieties of sphincter was that actual structural changes may have been brought about during the surgical procedure.

Bearing these studies in mind, it would seem that the personnel from the fields of radiology, surgery, and speech pathology, who are involved with the rehabilitation of the postlaryngectomized patient, need more specific information on the standard roentgenographic and cinefluorographic procedures available to them for evaluating postlaryngectomized physiology. For the best results from each individual surgical procedure, the following information is necessary: the best site for the pseudoglottis, the limits of subpseudoglottal pressures necessary for specific speech sounds, the surgical modification which will result in a more intelligible speech, the synchronization of pulmonary and phonic respiration, and the interrelationships of deglutition and phonation in postlaryngectomized speakers.

BASIC CINEFLUOROGRAPHIC DIAGNOSTIC TECHNIQUES APPLICABLE TO THE PHARYNX

The purpose of this article is to complement the above-mentioned publications by presenting useful examples of selected cinefluorographic (frame-by-frame) studies which will demonstrate the kinesiology of the soft palate and its related pharyngeal structures and their importance in deglutition and phonation.

The technique employed in our present study consists of 4 phases: (1) cephalometric filming, (2) cephalometric tracing analysis, (3) cinefluorographic filming during deglutition and phonation, and (4) cinefluorographic tracing analysis. The cephalometric equipment and filming procedures have been previously described.[13,25,26]

Cinefluorographic films were obtained with a 9 inch intensifier and a 35 mm. camera. The pulse stabilizer produced pulses of a 3 millisecond duration (24 pulses per second), while the 35 mm. camera (4 inch lens) was filming at 24 frames per second. Added beam filtration consisted of 1 mm. aluminum and 0.1 mm. copper, and the target table top distance was 20 inches below the table top. The dosimetry data taken during the study are recorded in Table I. The measurements were made while the patient was in position at 6 inches above the table top and calculated for a 20 per cent backscatter. The average filming time per examination was 20 to 30 seconds per patient.

Cinefluorograms of each subject were taken during deglutition (three swallows of barium microtrast) and phonation (vowels "ee" [i], "ah" [ɑ], "oo" [u]). In contrast to the fairly predictable physiology of swallowing, the oral and pharyngeal kinesiology changes for each phoneme (sound) being produced. A "cine" study of the three vowel sounds listed above, however, will provide the clinician with information about the patient's maximum and minimum phonation capabilities of all vowel sounds in isolation (Fig. 1). In order to assess the patient's continuous speech capabilities, it is important to have an understanding of the mechanism of consonantal sound production (Fig. 2), since all languages are composed of various vowel-consonant combination. One of the most useful consonantal sounds is "s" [s], particularly in the assessment of palatal incompetency patients. A short sentence, such as "*Ring the Bells and Sing*," provides a good sample of the patient's continuous speech pattern. Our cinefluorographic studies are made with the patient in a standing or sitting position. In order to ensure the proper magnification ratio between the cephalometric tracings of the still roentgenograms and the cinefluorographic roentgen-ray frames, a small steel rod, 5 inches in length with a 1 mm. notch each centimeter, was placed in the midline of the patient's mouth during the initial cinefluorographic filming. A Cine-Analyzer projector,* which enables the viewer to superimpose the cephalometric tracing over the selected cinefluorographic frame, is used for analysis of the cinefluorographic tracing analysis.[25]

Two cases were selected from the cephalometric-cinefluorographic study of 70 patients to demonstrate: (1) normal deglutition (Fig. 4); (2) deglutition in a palatal incompetency patient (Fig. 5); (3) normal phonation of [ɑ] (palatal competency, Fig. 6); and (4) palatal incompetency during phonation of [i] and [ɑ].

Very few differences can be noted by the viewer between the normal deglutition

Table I

CINEFLUOROGRAPHY DOSIMETRY

	kv.	ma.	Type Used	r per minute		Added Filter (Fig. 3)
				Air without Backscatter	Skin	
1.	65	20	Cinefluorography	0.85	1.0	No
2.	65	40	Cinefluorography	1.75	2.1	Yes
3.	65	0.2	Fluorography	0.22	0.27	No
4.	65	0.5	Fluorography	0.34	0.41	No

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* Model 710, Ralke Company, 849 North Highland Avenue, Los Angeles, California.

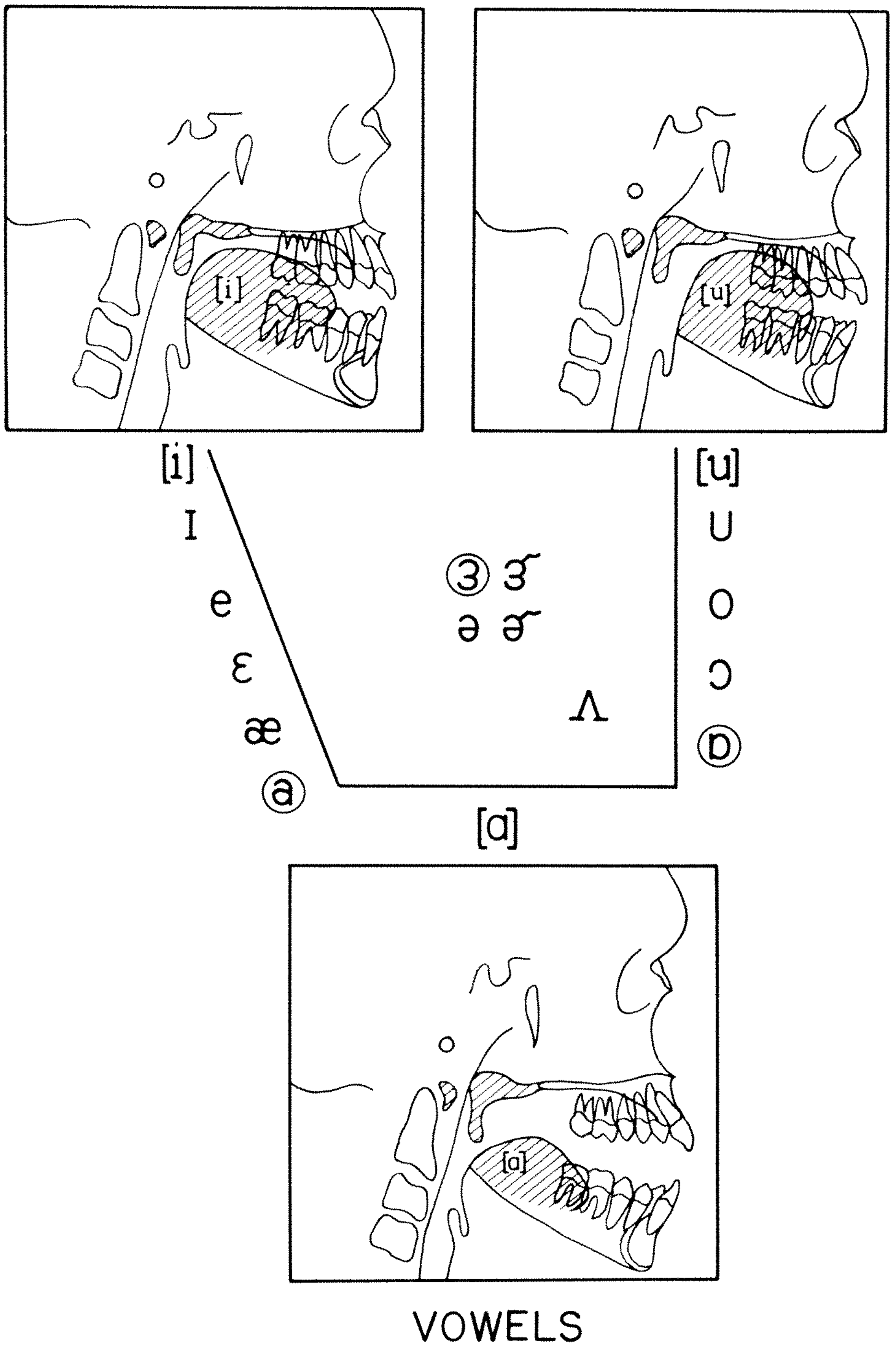
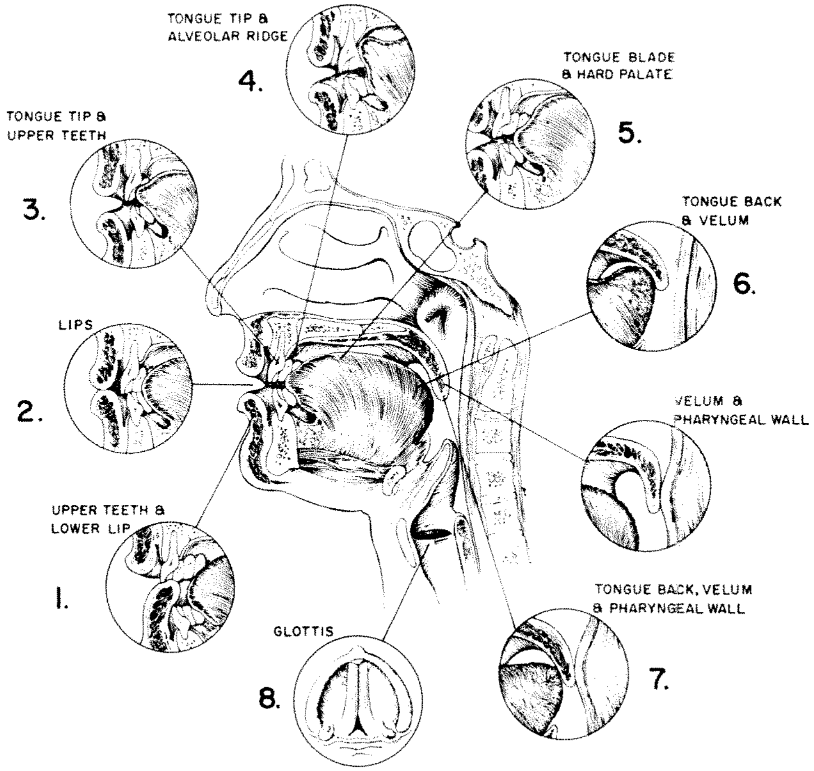


Fig. 1. Chart illustrating the common vowels. Uncircled vowels are common in American speech. The front vowels (left column) are produced with the tongue in the anterior portion of the mouth moving from a pattern noted in [i] to [ɑ]. The medial vowels are produced when the tongue is in a medial vertical position ([ɑ]). Back vowels (right column) are usually produced when the posterior dorsal aspect of the tongue is elevated toward the soft palate noted in [u] to [ɑ].

PHYSIOLOGICAL CLASSIFICATION OF CONSONANT SOUNDS



STRUCTURAL COMPONENTS OF THE ARTICULATORY VALVE	CLOSED VALVE		CONSTRICTED VALVE		VOICED NASALS
	VOICELESS	VOICED	VOICELESS	VOICED	
1. Labio-dental (upper teeth & lower lip)			"f" [f]	"v" [v]	
2. Bi-labial (lips)	"p" [p]	"b" [b]		"w" [w]	"m" [m]
3. Linguo-dental (tongue tip & upper teeth)			"th" [θ]	"th" [ð]	
4. Linguo-alveolar (tongue tip & alveolar ridge)	"t" [t]	"d" [d]	"s" [s]	"z" [z] "l" [l]	"n" [n]
5. Linguo-palatal (tongue blade & hard palate)	"ch" [tʃ]	"j" [dʒ]	"sh" [ʃ]	"zh" [ʒ] "r" [r] "y" [j]	
6. Linguo-velor (tongue back & velum)					"ng" [ŋ]
7. Linguo-velor-pharyngeal (tongue back, velum & pharyngeal wall)	"k" [k]	"g" [g]			
8. Glottal (glottis)			"h" [h]		

Fig. 2. Chart representing kinesiological positions for the oral and pharyngeal musculature during articulation of consonantal sounds. Areas 6 and 7 show tongue in an incorrect position in order to emphasize that the entire medial dorsal aspect of the tongue is normally found in contact with soft palate during production of sounds "ng," "k," and "g."

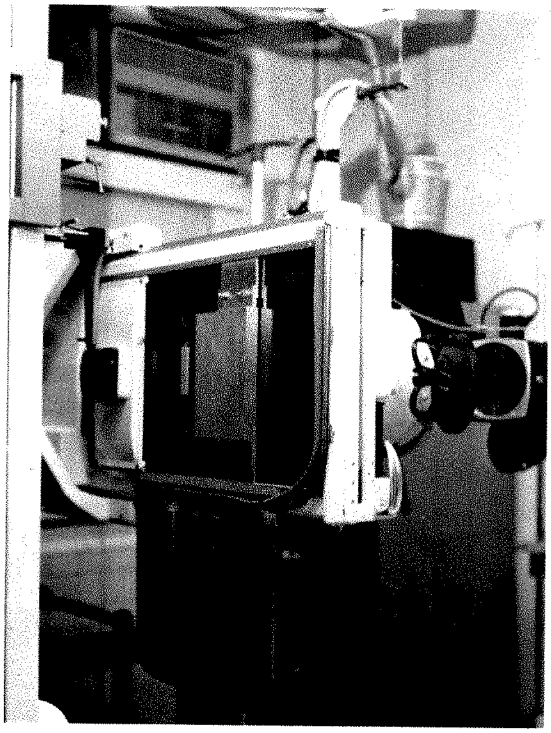


FIG. 3. Cinefluorographic wedge filter for 9 inch intensifier.

pattern (Fig. 4) and that of the patient with palatal incompetency deglutition pattern (Fig. 5). It has been previously reported by the authors[1] that the majority of palatal incompetency patients during a roentgenographic deglutition examination will demonstrate a compensatory (adequate) deglutition mechanism. Therefore, this technique should not be used as the only means of evaluating oral and pharyngeal components of the patient's speech mechanism.

It is the present opinion of the authors that information obtained of the degree of compensation of the pharyngeal sphincter demonstrated in palatal incompetency is very helpful in estimating the potential of the kinesiological limits of the patient's speech mechanism.[25] This information placed in the hands of a speech therapist skilled in speech rehabilitation for the cleft palate patient will necessarily reduce the hours of therapy.

It is of interest to note that frames 1 to 5 of Figure 5 clearly demonstrate a wide nasopharyngeal orifice as compared to frames 1 to 5 of Figure 4, which demonstrate the competent soft palate closure of the nasopharyngeal orifice. When one compares frames 6 to 10 of both Figures 4 and 5, it is of interest to note that in frames 6, 7, and 8 of Figure 5, the posterior aspect of the tongue is depressed to allow the bolus to move, seemingly by taking advantage of gravity, and at the same time it creates its own nasopharyngeal "seal," thus allowing for a minimal amount of nasopharyngeal regurgitation. This type of compensatory pattern is common in the majority of palatal incompetency patients. In the normal mechanism (frames 6–10, Fig. 4) the tongue and its associated lateral pharyngeal musculature (palatopharyngeus, etc.) "push" the bolus against the elevated adequate soft palate and the pharyngeal peristaltic action is initiated.

During phonation of the vowels (Fig. 6), the soft palate must be in contact with the posterior pharyngeal wall (superior constrictor), usually at the level, or slightly above the arch, of the atlas (*cf.* frames 1–5, Fig. 6). If this nasopharyngeal closure is not maintained, "hypernasal" vowel production will occur. This can be seen in frames 1A–5A and 1B–5B of Figure 6.

It has been noted that the immediate postoperative speech of patients recovering from the removal of tonsils and adenoids or pharyngeal flap procedures demonstrates denasality, primarily because the nasopharyngeal orifice is constantly closed by the edematous condition of the operative site.[1,25] Therefore, in order to obtain a true analysis of a patient's phonation potential, a postoperative period of at least 3 weeks should elapse before a speech analysis is requested.

SUMMARY

A brief review of the more recent advances in cinefluorographic filming techniques for studying the physiology of the cranial and pharyngeal areas is given. The basic diagnostic techniques for cinefluorographic examination of the pharyngeal regions during deglutition and phonation

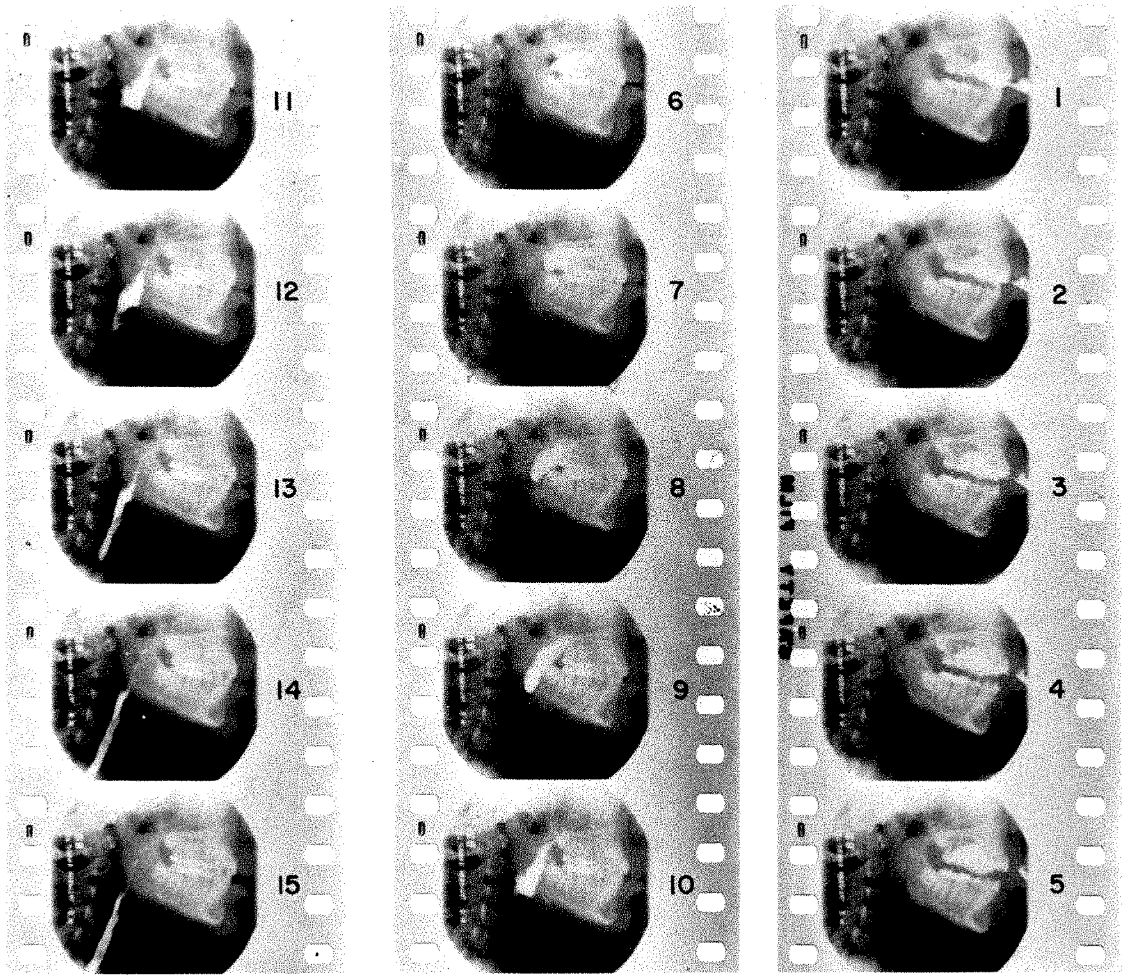


FIG. 4. Normal deglutition pattern of a 15 year-old female.
(*Please turn the page for Fig. 5 and 6*)

are outlined. Two cases are presented to demonstrate these techniques. Palatopharyngeal competency and incompetency are briefly discussed.

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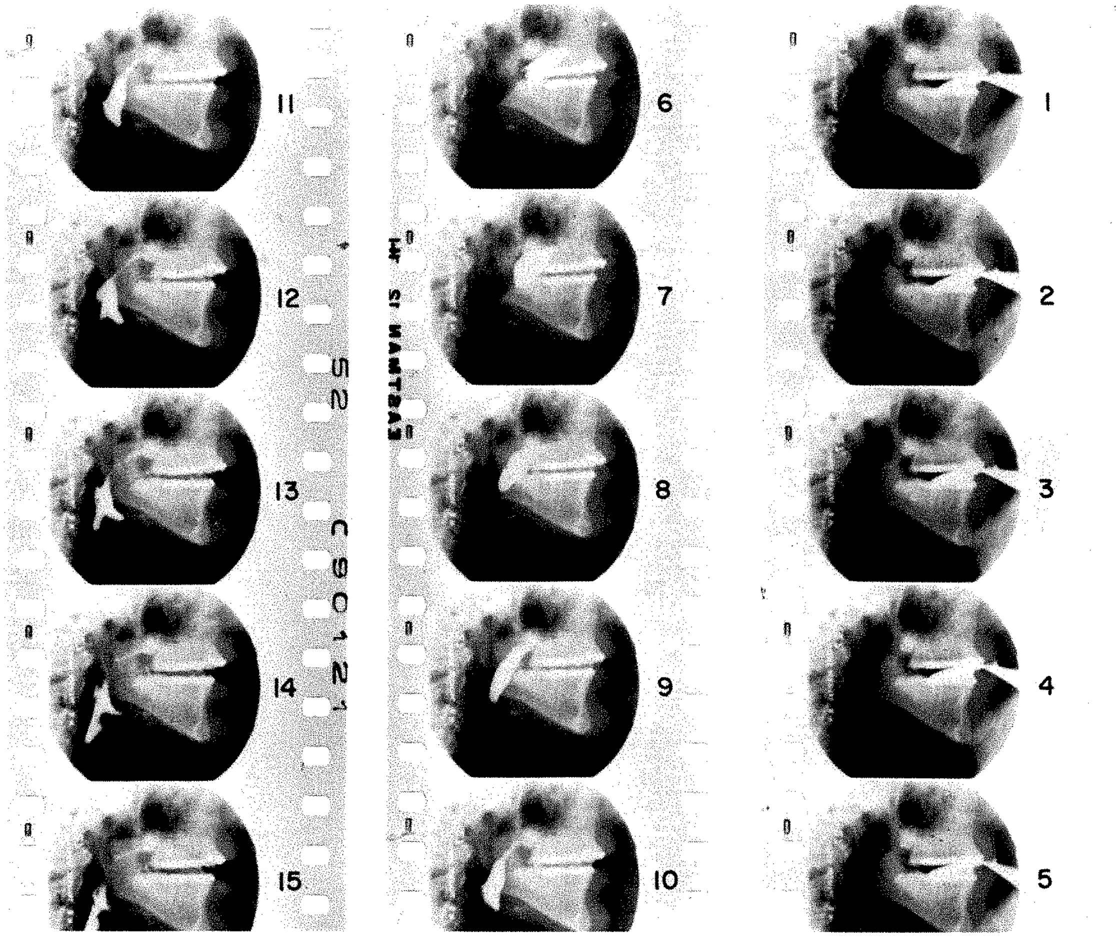

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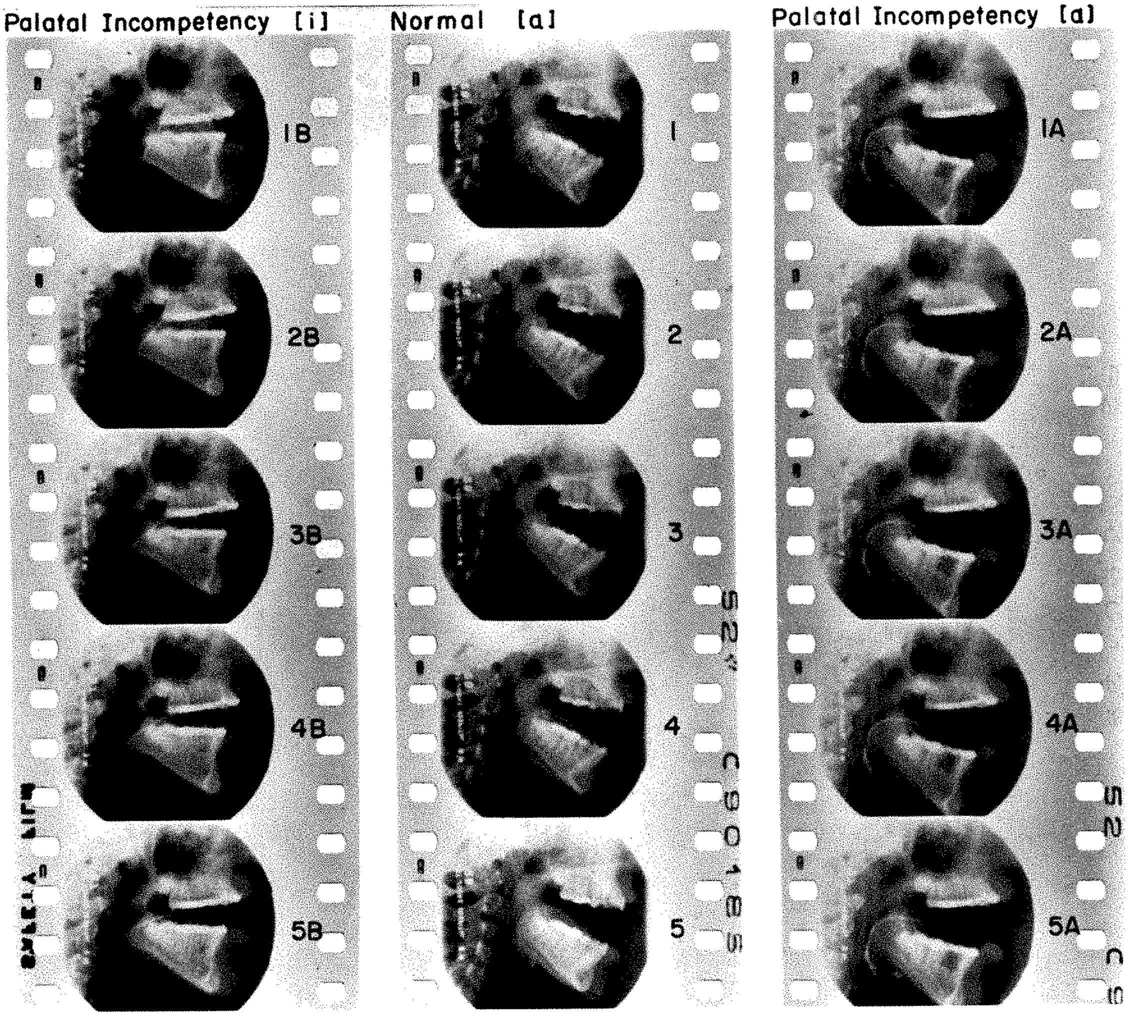

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COMBINED PRESSURE TRACINGS AND CINE-ROENTGENOGRAPHY OF HOLLOW MUSCULAR VISCERA*

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RECORDINGS of intraluminal pressure and cineroentgenography are two important methods for studying the function of the hollow muscular viscera. Both methods, however, have obvious limitations.

Pressure tracings obtained from multiple adjacent levels can show peristaltic waves as they progress along the organs and give reliable data on the dynamic values. Pressure tracings, however, do not register the motions of visceral contents.

On the other hand, cineroentgenography reveals the changes of the outline of a viscus and of the movement of its contents but is unable to furnish information on the complex muscular contractions.

Since the two methods are incomplete when employed separately, it appeared that it might prove useful to employ them together so that the data obtained from one could be correlated with those from the other.

By filling the organ with radiopaque fluid, using catheters and transducers and employing cineroentgenography, simultaneous records of the pressure changes and of the movement of visceral contents were obtained. To register intraluminal pressures a recorder was connected to the pressure transducers and to catheters with multiple channels so that pressures were usually taken 5, 10 and 15 cm. from the tips of the catheters. These catheters were positioned under fluoroscopic control. The cineroentgenographic equipment consisted of a conventional cine-television unit.

The synchronization of the two recordings was assured by a time marker registering on the cine film as well as on the recording drums.

These combined pressure-cineroentgenographic studies have been carried out at our institution on humans since 1956, first on the esophagus and then on the uterus and on the gastrointestinal and urinary tracts. Our results have been fully reported elsewhere.[1–4,6,7,15] On the basis of this experience, it was thought of interest to discuss briefly the problem of correlating pressure tracings with the cineroentgenographic films as well as the value of such a combined procedure.

Basically, cineroentgenograms and pressure tracings represent very different types of records. Pressure tracings are quantitative registrations of changes in time and amounts. Both items can be accurately calibrated and measured, and single or multiple events can be recorded and studied with care. Cineroentgenograms, on the other hand, are qualitative records of fleeting events and, when projected, only the memory of the observer can compare the current event with the preceding ones.

From a practical standpoint, therefore, a correlation between pressure tracings and cineroentgenograms can be accomplished only by studying the cine frames one by one after the frames have been accurately located in time with respect to the events shown by the pressure recording. This is facilitated by pasting copies of the proper cine frames at the proper points of the pressure tracings (Fig. 1).

The data furnished by such combined recordings have helped us to study many motor events and especially those which

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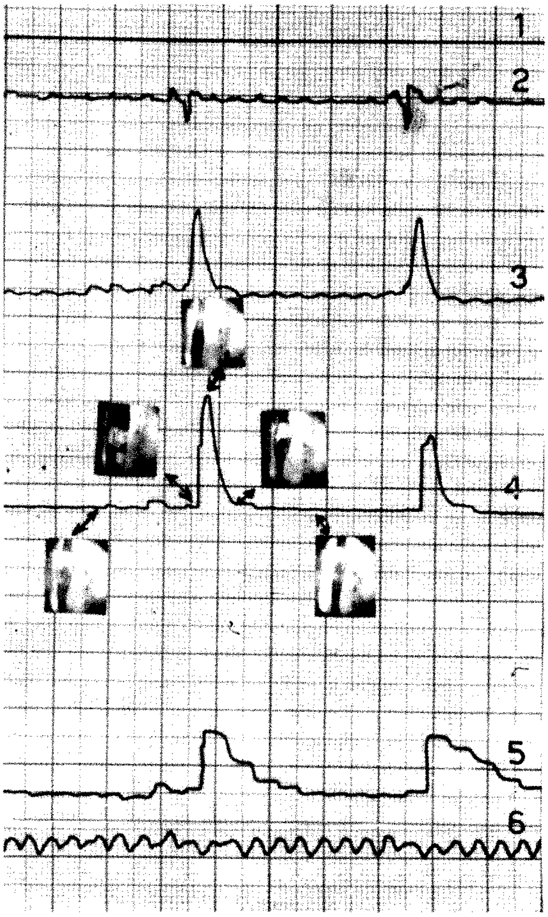

Fig. 1. Combined study of esophagus: tracing 1, synchronization; tracing 2, signal of swallowing; tracing 3, upper esophagus; tracing 4, middle esophagus; tracing 5, lower esophagus; and tracing 6, respiration.

occur at transition points such as the pharyngoesophageal, esophagogastric and pyloroduodenal junctions. The knowledge of the pressure existing above and below such sphincteric regions, coupled with the passage of opaque material, offers an opportunity for physiologic observation which cannot be duplicated by other methods at the present time.

CONCLUSION

We believe that a better understanding of mechanical factors involved in the motion of the contents of the hollow viscera may be gained by the simultaneous use of cineroentgenography and pressure recordings. We also hope that, occasionally, this combined method may be of clinical value in the detection of those infiltrative lesions of hollow viscera which so often escape our most conscientious diagnostic efforts.

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The practical work in this study was carried out by A. Curatolo and E. Salomoni.

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ESOPHAGEAL REFLUX

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THERE are many references in the literature to the importance of esophageal reflux but far fewer to the frequency with which it occurs. Johnstone[5] has repeatedly emphasized the importance of esophageal reflux. In 1955 he reported that the presence of reflux was a vital observation, because with it there was a 75 per cent chance that the patient had esophagitis whether or not a hiatus hernia was present.

Cross and associates,[3] in 1959, reported on 130 patients with reflux. Of these, 27 had no demonstrable hiatus hernia.

Conway-Hughes,[2] in 1956, tested 435 patients for reflux and found 11.5 per cent to have free reflux and 8.8 per cent to have forced reflux; *i.e.*, reflux caused by applying additional abdominal pressure. There was no correlation with any lesion except hiatus hernia. He used the Trendelenburg position with and without abdominal pressure.

Meiss and associates[6] found that reflux esophagitis in the dog could not be produced by displacing the lower esophageal sphincter up into the mediastinum if the hiatus was not tight enough to cause obstruction of the stomach pouch. They also found that the mucosal rosette, the angle of insertion of the esophagus into the stomach, and the muscle of the diaphragm around the hiatus had no relationship to reflux esophagitis. In fact, the only experimental surgery that consistently produced reflux esophagitis was removal of the lower esophageal sphincter itself. The fact that simple displacement of the lower esophageal sphincter up into the chest, that is, hiatus hernia, is often associated with esophagitis in humans, they speculated, is due to the fact that the hiatus is often tight enough to cause partial obstruction of the supradiaphragmatic pouch of stomach. This was also emphasized by Effler and Groves.[4]

With cine recording and image amplification, the motor activity of the lower part of the esophagus along with fleeting changes such as esophageal reflux can be studied readily. Using this method, we have attempted to find the incidence of gastroesophageal reflux and to study its relationship to hiatus hernia.

MATERIAL AND METHODS

The patients examined were those referred to the section of diagnostic roentgenology for regular clinical stomach examinations. These patients had a wide variety of complaints which could reasonably be attributed to upper gastrointestinal disease. They were ambulatory outpatients without previous operations on the esophagus or the stomach.

After the conventional upper gastrointestinal fluoroscopy and filming, the patients were examined by a second examiner in a separate room. The second examination was completely independent of the first. It was done only to evaluate the lower portion of the esophagus for hiatus hernia and reflux.

During the first examination, each patient received approximately 9 ounces of barium suspension. For the second examination the patient was given a teaspoonful of thick barium to hold in his mouth. He was then instructed to lie on the table on his back and to roll up slightly on his left side. In this position he was instructed to swallow the thick barium; at the same time the region of the lower part of the esophagus and the esophagogastric junction was viewed on the image-intensifier screen. Any obvious or questionable evidence of esophageal hiatus hernia or reflux was recorded on cine film and reviewed later. This second examination took less than 3 minutes per patient on the average.

The 16 mm. cine strips that were exposed in all positive and borderline cases were reviewed several times by the authors. A punch card was made out for each patient and several specific findings were tabulated, as follows:

1. Sex and age

From cinefluorography

2. Esophageal reflux
3. Esophageal hiatus hernia (small, medium, and large)
4. Motor incoordination of the lower portion of esophagus

From routine fluoroscopy and filming

5. Esophageal hiatus hernia
6. Duodenal ulcer
7. Special films for demonstration of hiatus hernia

These findings were then evaluated to see if a correlation existed between reflux and any of the other factors.

As a check, approximately 50 cine examinations were reviewed at a later date without knowledge of the original evaluation. There was almost complete agreement with our original diagnoses.

RESULTS

Gastroesophageal reflux was demonstrated in 10.2 per cent (51/500) of all patients. Of the 51 patients with reflux, 98 per cent (50/51) had esophageal hiatus hernia, 18 per cent (9/51) had duodenal ulcer, and 20 per cent (10/51) had motor incoordination of the esophagus.

The only significant correlation with reflux was the finding of esophageal hiatus hernia. Hiatus hernia occurred in 40.6 per cent (203/500) of all patients, but it was present in 98 per cent of patients with reflux.

Duodenal ulcer occurred in 12 per cent (60/500) and motor incoordination in 8 per cent (39/500) of all patients. Table I shows the frequency and correlation of all factors studied.

COMMENT

Normally, the esophageal peristaltic wave progresses in an orderly manner down the esophagus and through the esophageal hiatus to the gastroesophageal junction (Fig. 1). The diagnosis of reflux was made when barium in the stomach regurgitated back into the esophagus (Fig. 2 and 3). The diagnosis of esophageal hiatus hernia was

TABLE I

INCIDENCE AND ASSOCIATIONS OF GASTROESOPHAGEAL REFLUX IN 500 ROUTINE EXAMINATIONS*

	Total Patients	Cinefluorography							Routine Examination		
		Reflux	H.H.				M.I.				
			Total	Small	Medium	Large			H.H.	D.U.	3DH
Reflux (cine)	51	51	50	42	8	0	10		18	9	27
H.H. (cine)	203	50	203	180	19	4	30		91	28	114
Small	180	42	180	180	—	—	25		70	24	93
Medium	19	8	19	—	19	—	5		17	3	17
Large	4	0	4	—	—	4	0		4	1	4
M.I. (cine)	39	10	30	25	5	0	39		14	9	22
H.H. (routine)	91	18	91	70	17	4	14		91	12	82
D.U. (routine)	60	9	28	24	3	1	9		12	60	21
3DH roentgenograms (routine)	175	27	114	93	17	4	22		82	21	175

* More significant numbers are underlined. H.H.=hiatus hernia; M.I.=motor incoordination of the esophagus; reflux=gastroesophageal reflux; D.U.=duodenal ulcer; 3DH=roentgenograms obtained during the routine examination with the patient swallowing to demonstrate hiatus hernia.

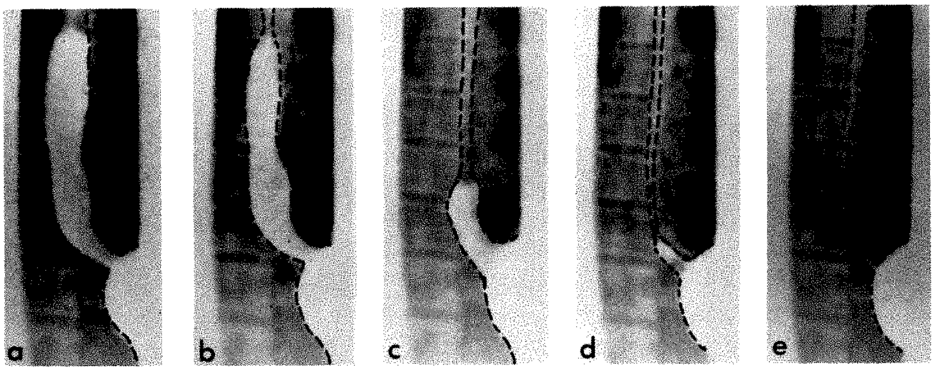

Fig. 1. Normal esophagus and stomach. Normally, peristaltic wave proceeds down esophagus through lower esophageal sphincter which lies in esophageal hiatus of diaphragm and stops at esophagogastric junction. Lower esophageal sphincter regains its resting tone; no reflux occurs.

made on the basis of the following features: 1. Enough of the stomach was above the diaphragm so that it was easily recognized by its gross appearance (Fig. 2). 2. Careful observation of the terminal esophageal peristaltic wave revealed that it stopped above the esophageal hiatus. In almost all cases, the lower limit of the peristaltic contraction corresponded to a visible lower esophageal ring, which was assumed to be the mucosal esophagogastric junction (Fig. 3).

Motor incoordination was diagnosed when disordered nonperistaltic motor activity of the lower portion of the esophagus occurred and gave rise to the characteristic pattern sometimes known as curling, tertiary contractions, corkscrew, rosary, or rippling.

The finding of duodenal ulcer was included for all patients who had a crater or deformity of the duodenal cap on the regular clinical portion of the examination.

If any significance is to be attached to the demonstration of reflux and hiatus hernia, they should probably not be demonstrated by grossly unphysiologic maneuvers. For this reason we chose not to use any of the many techniques which artificially raise the intra-abdominal pressure above that which the patient might ordinarily be expected to experience.

The lower portion of the esophagus was observed in the supine position (LPO) with the patient swallowing, and in some cases with breath-holding in inspiration. The Trendelenburg position was also used routinely during the initial portion of the study, but it was later abandoned because it did not seem to increase the incidence of

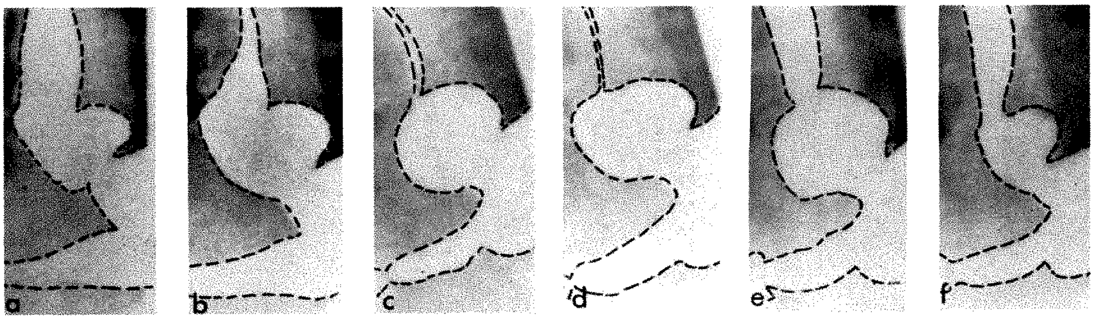

Fig. 2. Moderate hiatus hernia with reflux. Peristaltic wave can be seen to pass down esophagus and stop at esophagogastric junction (frames a, b, c, and d). Moderate sized supradiaphragmatic pouch of stomach is filled, and it does not contract in association with esophageal peristalsis (frame d). When pressure in hernial pouch is greater than that of resting pressure in lower esophageal sphincter, reflux occurs (frames e and f).

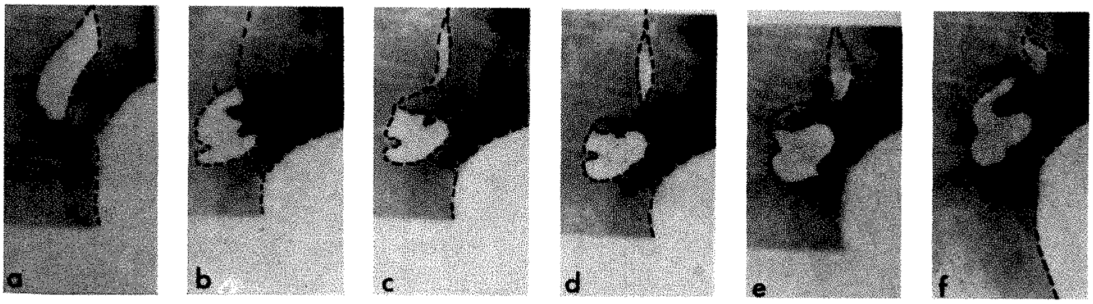

FIG. 3. Small hiatus hernia with reflux. Peristaltic wave can be seen to proceed down esophagus and end at a small hiatus hernia. Lower esophageal ring is demonstrated at terminal phases of peristaltic wave (frames b, c, and d). Subsequent swallow relaxes lower esophageal sphincter, and reflux occurs (frame f). Typical configuration of lower esophageal sphincter is seen in frames c, d, and e.

demonstrable hernia or reflux. Experimental work done here with simultaneous cinefluorography and multiple intraluminal esophageal pressure recordings by one of the authors (H.C.C.) supports our observation that the motor sequence and termination of the esophageal peristaltic wave as seen in the supine position helps define the lower esophageal sphincter and hence the gastroesophageal junction. The lower esophageal sphincter appears to have a characteristic contraction and appearance in most patients, even in the absence of a persistent lower esophageal ring.

Esophageal reflux, for the most part, consisted of the regurgitation of stomach contents from a small supradiaphragmatic pouch into the esophagus during or at the end of a peristaltic contraction. Few of these patients had massive reflux of stomach contents from below the diaphragm into the esophagus. It was felt, however, that the chronic, less dramatic, reflux of the stomach contents from a small pouch of herniated stomach into the esophagus might be of some significance.

Code and associates[1] have shown that, normally, the resting pressures in the stomach are always higher than those of the esophagus. There is, however, a short region of increased pressure which forms a barrier between the stomach and the esophagus. The pressure in this zone is higher than the pressure in the esophagus and stomach, in any position including the Trendelenburg position, and thereby prevents gastroesophageal reflux. This area, the lower esophageal sphincter, relaxes when a swallow is initiated. The onset of pressure decrease occurs early in the swallowing sequence, and contraction does not occur until the peristaltic wave passes through the sphincter. The pressure wave enters the sphincteric zone and closes the sphincter and again establishes the necessary pressure barrier between the esophagus and the stomach.

Some patients with esophageal hiatus hernia exhibit reflux during that vulnerable period after the lower esophageal sphincter has relaxed in response to swallowing, and before the peristaltic wave has arrived at the sphincter and the pressure barrier has again been established (Fig. 3). Also, any patient with a small hiatus hernia may have reflux if, during swallowing, the esophageal hiatus is closed more tightly than the terminal esophageal peristaltic wave, thereby allowing any contents of the hernial pouch to regurgitate back into the lower portion of the esophagus. Contents of the hernial pouch could then more easily regurgitate into the esophagus than proceed through the hiatus. In our study the only significant lesion found to be correlated with reflux was hiatus hernia.

In recent years, refined techniques have shown that the incidence of hiatus hernia is much higher than was formerly reported. In fact, in some series the incidence has exceeded 50 per cent.[7] Hiatus hernia may be a common degenerative change in older people, similar to degenerative arthritis; however, the question must be raised whether a "small esophageal hiatal hernia" is, in fact, an abnormality. It would be helpful if some method could be found to sort out the more important forms of hiatus hernia. Thus, those that can be demonstrated without the use of unusual abdominal pressure and those that are associated with reflux may be more significant. This remains to be proved, however, and we did not correlate the roentgenologic findings in our cases with the clinical features.

SUMMARY AND CONCLUSIONS

Five hundred consecutive patients who were undergoing routine upper gastrointestinal roentgenologic examination were given an independent examination for esophageal hiatal hernia and gastroesophageal reflux. Special care was taken to examine the lower esophageal region by means of cinefluorography with thick barium in the recumbent position.

The incidence of reflux was found to be 10.2 per cent in all patients. Of 51 patients exhibiting reflux from stomach to esophagus, 50 had esophageal hiatal hernia. Although the difference was not statistically significant, reflux was noted to occur more commonly with the small than with the large hiatus hernia. Of 203 patients with esophageal hiatus hernia demonstrated by cineroentgenography, 50 (24 per cent) had reflux.

During roentgenographic examination we have observed 3 mechanisms of gastroesophageal reflux resulting from relatively incompetent lower esophageal sphincter and associated with hiatus hernia:

1. Reflux occurs when there is reflex relaxation of the lower esophageal sphincter subsequent to swallowing in patients with hiatus hernia; contents of the hernial pouch regurgitate up into the esophagus before being swept back into the stomach by the ensuing esophageal peristaltic wave.

2. When, in the presence of a small hiatus hernia, the hiatus is closed more tightly than the oncoming esophageal terminal peristaltic wave, contents of the hernial pouch regurgitate back through the terminal peristaltic contraction.

3. In the presence of a hiatus hernia, when the esophageal hiatus is widely patent and any increase in the intra-abdominal pressure is easily transmitted through the hiatus into the supradiaphragmatic pouch, reflux ensues. In this case, unusually high pressures are challenging the lower esophageal sphincter at a time when it is displaced out of the esophageal hiatus and is relatively unsupported.

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CONGENITAL ESOPHAGEAL STRICTURES

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CONGENITAL esophageal strictures are uncommon and the term "congenital esophageal web" has been used to describe them. The term "web" implies an incomplete diaphragm of developmental origin compromising the esophageal lumen. Sometimes longer areas of esophageal narrowing occur independently or in association with webs. These may be referred to as areas of segmental stenosis and may be up to 10 cm. in length.[6]

Historically, the first mention of congenital esophageal strictures dates back to a description by Blasius[3] in 1674. Prior to 1928, Beatty could find only 50 cases of congenital esophageal stricture in the entire world literature, but the authenticity of many of these cases could be questioned.[2]

The first well-documented cases of congenital esophageal stricture in the American literature were reported by Rotch in 1913.[11] Of his 3 cases, 2 were typical clinically and were verified by roentgenographic studies.

The largest single series was that of Gross[4] who presented 38 cases of congenital esophageal stricture. In his cases, 75 per cent of the lesions were detected by the time the patient was 2 years of age. In approximately 50 per cent, the obstructing lesion was in the middle third of the esophagus, and in 25 per cent, the lesion was in the lower third.

The purpose of the present author is to present 6 cases as well as a discussion of congenital esophageal strictures in children.

ETIOLOGY

The etiology of congenital esophageal strictures is more evident if one considers the embryology involved. The respiratory tree arises from a ventral evagination in that portion of the esophagus just caudal to the pharyngeal pouches. Normally, by the 4–5 mm. fetal stage, the trachea and esophagus have completely separated.[8] Esophageal atresia with tracheo-esophageal fistulas and esophageal strictures caused by tracheobronchial tree remnants arise during this period of development.[9,10] The formation of esophageal strictures occurs after the 13 mm. fetal stage when vacuoles are formed in the esophageal epithelium.[7] These vacuoles are first noted immediately above the level of the tracheal bifurcation but by the 16 mm. fetal stage, they are present throughout the esophageal epithelium. The vacuoles occur on all walls. As the vacuoles enlarge, the main esophageal lumen is considerably narrowed, especially in that portion of the esophagus below the tracheal bifurcation, but it is never completely obliterated. The final purpose of the vacuolization stage is enlargement of the esophageal lumen. Figure 1, *A* and *B* shows the complex lattice work during this vacuolization stage. By the time the fetus has

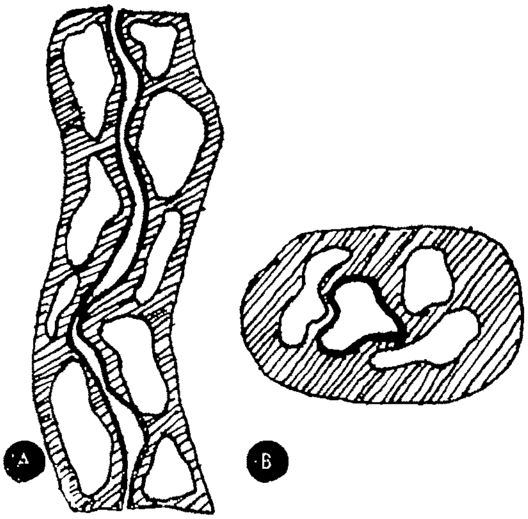

FIG. 1. (*A*) Sketch of longitudinal section through the developing esophagus near the 20 mm. fetal stage. Dark lines outline main esophageal lumen. Clear ovoid areas are the vacuoles in the esophageal epithelium. (*B*) Sketch of cross section through the same developing esophagus as in *A*. Again dark line outlines main esophageal lumen.

reached the 72 mm. stage, the vacuoles have coalesced and the 4 primary longitudinal folds of the esophagus are formed. This failure of coalescence is thought to be responsible for esophageal stricture formation.

HISTOLOGY

In most instances, congenital esophageal strictures are adequately treated by nonsurgical methods; hence, histologic studies of these lesions are rare. Recently, Adler[1] reported a congenital esophageal stricture presenting as an incomplete diaphragm (web). Histologically, the web was composed only of a mucous membrane with the muscular layers of the esophagus uninvolved. This latter finding would seem to substantiate the vacuolization theory of origin of congenital esophageal strictures.

SYMPTOMATOLOGY

Classically, these children are "spitters" but may be asymptomatic until the time solid food is added to the diet. The vomitus is usually comprised in part of undigested food.

Not infrequently, an impacted esophageal foreign body first draws attention to the lesion. Any smooth foreign body which is able to pass through the level of the cricopharyngeus muscle will normally pass into the stomach. Failure to do so should strongly suggest the presence of either a congenital or an acquired esophageal stricture and should prompt a careful roentgenologic search for such a causative lesion.

ROENTGENOLOGY

Many children referred to us for roentgenologic study of the esophagus are too young to cooperate during the examination. Children under 3 years of age are usually immobilized and after a small radiopaque French catheter is introduced into the esophagus, contrast medium is injected by the technician on the request of the examiner. Thus, the radiologist is in complete control at all times and can perform an unhurried and thorough examination. Spot roentgenograms of the esophagus are routinely obtained when it is well distended with the contrast material.

The 3 congenital types of lesions that may be identified roentgenographically are:(1) an independently occurring esophageal web, (2) an area of segmental stenosis, and (3) an esophageal web occurring in association with an area of segmental stenosis. Usually, the proximal esophagus above the congenital stricture will be dilated while the esophagus below the lesion is of normal caliber.

TREATMENT

The vast majority of cases can be adequately treated by esophagoscopy with dilatation of the narrowed segments. When lesions are encountered that are unyielding to instrumentation, the possibility of this stricture being caused by tracheobronchial tree remnants must be considered.[9,10] Because of the cartilaginous ring that is frequently associated with these lesions, surgical resection is usually necessary.

REPORT OF CASES

CASE I. A 9 month old white male was admitted because of intermittent vomiting since 6 weeks of age, at which time solids had been started. Undigested food was frequently noted in the vomitus. Esophagrams (Fig. 2, *A* and *B*) showed a congenital esophageal web near the junction of the middle and distal thirds of the esophagus with an area of segmental stenosis distally. The esophagus was subsequently enlarged to accommodate a No. 22 French dilator.

CASE II. A 10 year old white male was admitted with a history of postprandial emesis following the introduction of solids into his diet during infancy. He was first seen at 2 years of age, when a roentgenogram showed a congenital esophageal stricture. No specific treatment was given at that time. The patient was currently admitted to our hospital for definitive treatment. An esophagram (Fig. 3) demonstrated a congenital esophageal web in the middle third of the esophagus with an area of segmental stenosis distally. The esophagus was

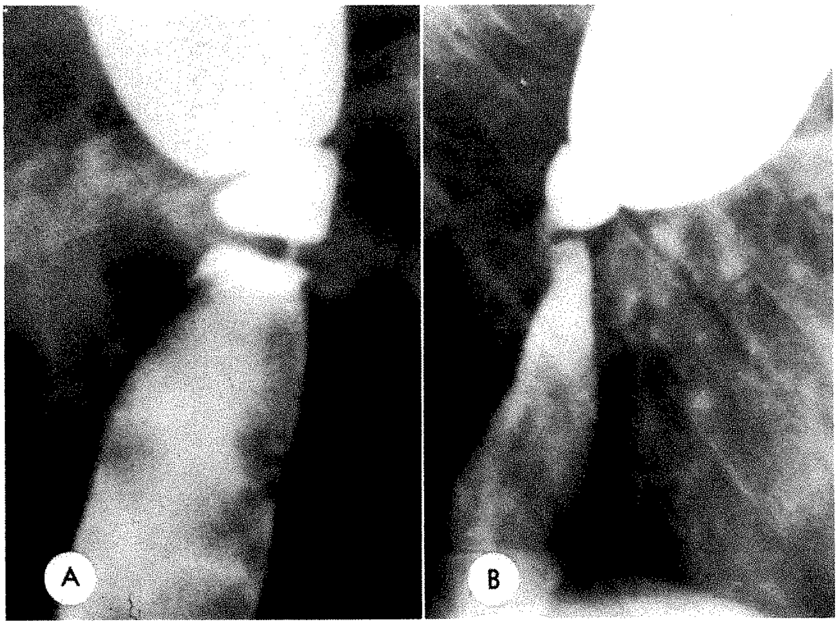

FIG. 2. Case I. (*A*) Esophagram showing a congenital web near the junction of middle and distal thirds of the esophagus with a more significant area of segmental stenosis distally. (*B*) Later view of same area.

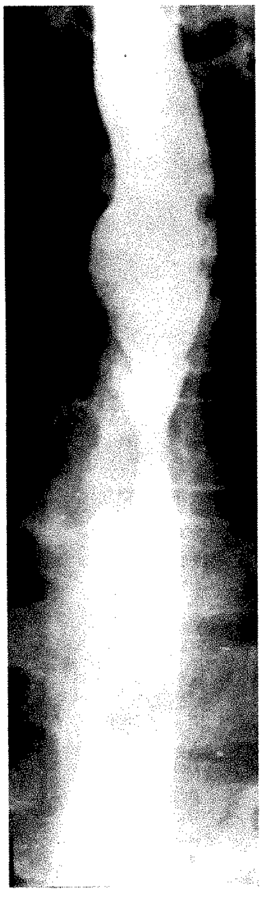

FIG. 3. Case II. Esophagram demonstrating a small congenital web in the middle third of the esophagus with an area of stenosis distally.

dilated to accommodate a No. 33 Plummer sound.

CASE III. An 11 month old white male was admitted to the hospital. He had always been a "spitter" but this had become worse after solids had been added to his diet. The vomitus consisted largely of undigested food. An esophagram (Fig. 4) revealed an area of segmental stenosis at the junction of the middle and distal thirds of the esophagus. The esophagus was enlarged to accommodate a No. 24 French dilator.

CASE IV. A 16 month old white male was admitted because of persistent emesis of undigested food containing large amounts of mucus. The patient had always been a "spitter" but during the past 7 months this had become worse. An esophagram (Fig. 5) showed a congenital esophageal web in the middle third of the esophagus with an area of segmental stenosis at the junction of the middle and distal thirds. The esophagus was enlarged to accommodate a No. 16 French dilator.

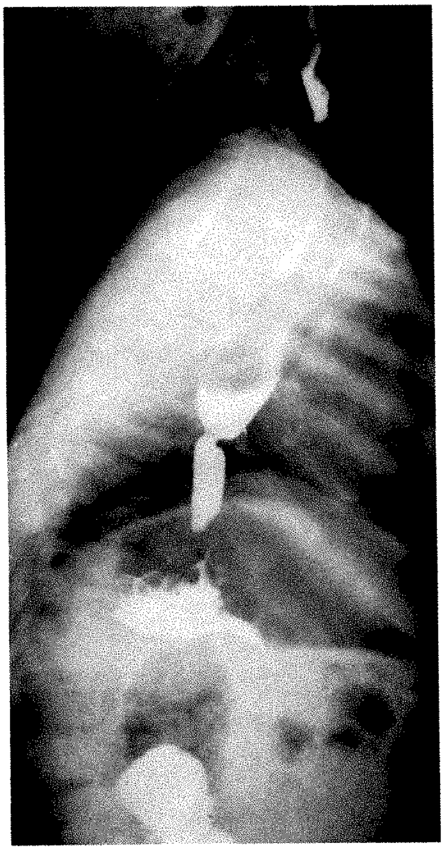

Fig. 4. Case III. Lateral esophagram showing a congenital area of segmental stenosis.

Case v. An 8 month old white male was admitted because of difficulty in swallowing solids. He frequently regurgitated undigested food and large amounts of mucus. An esophagram (Fig. 6) showed a congenital esophageal stricture near the junction of the middle and distal thirds of the esophagus. During esophagoscopy an infant-size esophagoscope could not be passed through the stenotic area, but a 3.5 mm. bronchoscope was successfully passed through the narrowed zone.

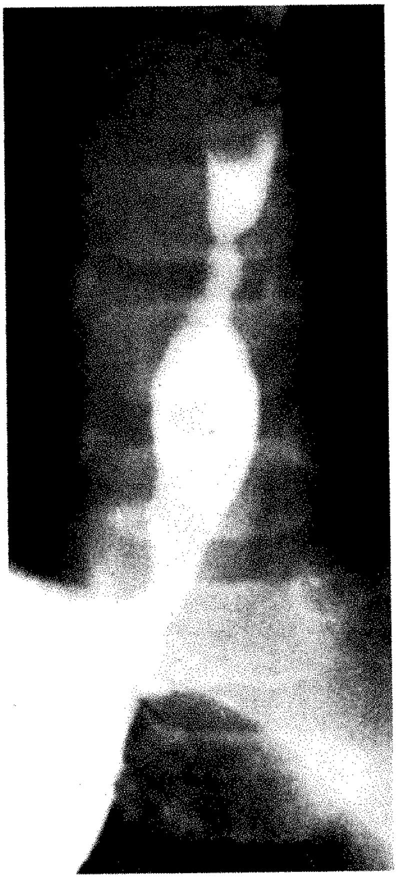

Fig. 6. Case v. Esophagram showing a congenital area of segmental stenosis near the junction of the middle and distal thirds of the esophagus.

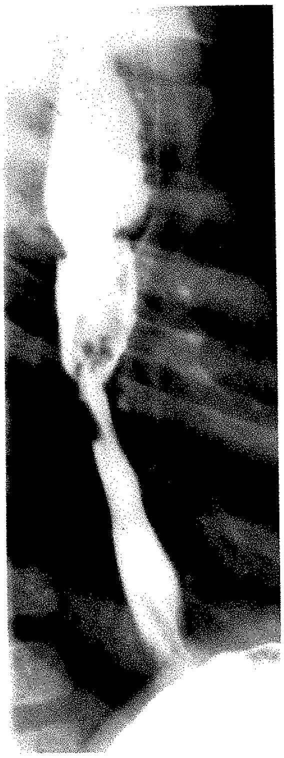

Fig. 5. Case iv. Esophagram revealing a congenital web in the middle third of the esophagus with a more significant area of segmental stenosis near the junction of the middle and distal thirds.

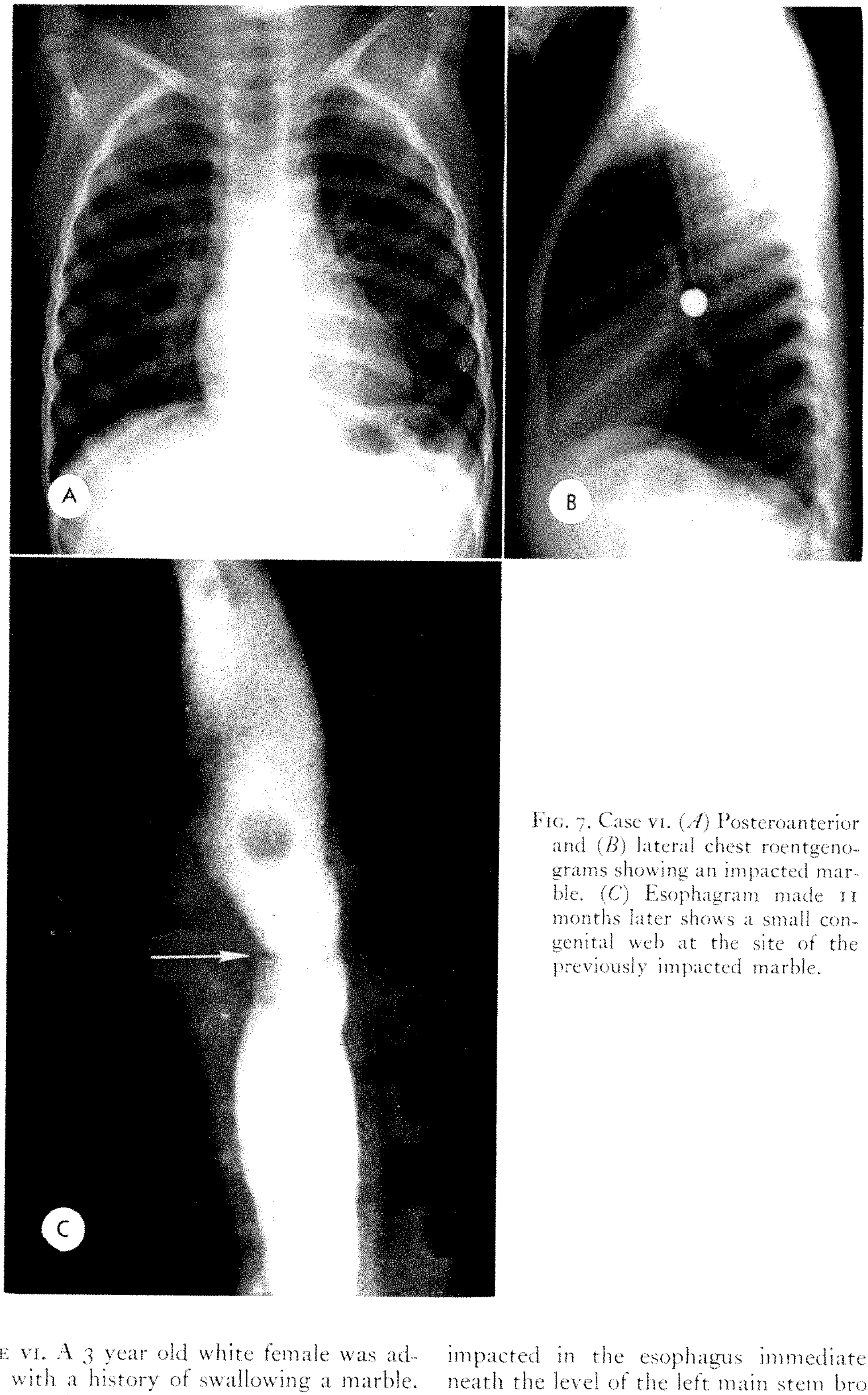

Fig. 7. Case vi. (*A*) Posteroanterior and (*B*) lateral chest roentgenograms showing an impacted marble. (*C*) Esophagram made 11 months later shows a small congenital web at the site of the previously impacted marble.

Case vi. A 3 year old white female was admitted with a history of swallowing a marble. Posteroanterior and lateral roentgenograms of the chest (Fig. 7, *A* and *B*) showed the marble impacted in the esophagus immediately beneath the level of the left main stem bronchus. An esophagram was not done at this time. Esophagoscopy was performed and the marble was pushed into the stomach as it was too tightly impacted to be removed through the esophagoscope. Eleven months later the patient was again admitted with emesis of undigested food. An esophagram (Fig. 7*C*) revealed a congenital esophageal stricture near the junction of the middle and distal thirds of the esophagus. An infant-size esophagoscope was subsequently passed through the stricture.

DISCUSSION

The age of discovery in our cases varied from 8 months to 36 months. In all of our cases the lesion was located near the junction of the middle and distal thirds of the esophagus. While congenital esophageal webs and areas of segmental stenosis have been previously described by other authors, I am aware of only one other report of the two lesions occurring in combination.[5] One possible explanation for this disparity is that the esophageal web is frequently small and is usually overshadowed by the more significant area of segmental stenosis immediately distal to the web. Unless a careful roentgenologic examination is performed in the involved area, the esophageal web could be easily overlooked. In 3 of our cases, this combination occurred. A more proximal web with an area of segmental stenosis was present immediately distal to the web. All of our cases were successfully treated by dilatation.

The findings in our cases, and a consideration of the embryology involved, would suggest that the webs and areas of segmental stenosis are probably different manifestations of the same developmental error, namely, failure of the epithelial vacuoles to coalesce.

CONCLUSION

1. Six cases of congenital esophageal stricture are presented; in each instance, the lesion was located near the junction of the middle and distal thirds of the esophagus.

2. In 3 of the cases, an esophageal web and an area of segmental stenosis were found in combination. This finding would indicate that webs and areas of segmental stenosis occur not only more frequently together than heretofore reported, but also represent different manifestations of the same developmental error.

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ESOPHAGEAL VARICES IN SUPERIOR VENA CAVAL OBSTRUCTION*

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ESOPHAGEAL varices caused by obstruction of the superior vena cava is an unusual and little known entity. We have accepted the term "down hill varices" to describe this condition because the direction of flow is the reverse of that in the varices of portal hypertension.

Recently, we have had an opportunity to study 4 patients with this entity. The purpose of this paper is to emphasize the mode of development and direction of flow in the mediastinal collateral venous pathways that may occur after superior vena caval obstruction.

REPORT OF CASES

CASE I. G.S., a 75 year old Negro male admitted because of carotid artery insufficiency, was found on physical examination to have dilated veins in the neck and over the chest and abdomen. The latter filled from above. The patient had noted these veins for the past 5 years. An episode of "pneumonia" 10 years previously was the only prior illness. He neither smoked nor drank alcoholic beverages. There were no symptoms referable to the chest or gastrointestinal tract. The liver and spleen were not palpable.

Venous pressure in the right antecubital vein was elevated to 184 mm. H_2O. Arm to tongue circulation time (decholin) was 28 seconds. The tuberculin skin test (second strength PPD) was negative, but the histoplasmin was weakly positive.

Laboratory Data. Routine blood studies and urinalysis were normal. Liver function studies including serum proteins and A/G ratio, serum bilirubin, alkaline phosphatase, serum transaminase, thymol turbidity, cephalin flocculation, and bromsulphalein retention were normal. Seventy-two hour sputum concentrates for acid-fast organisms were negative on smear and culture.

Bronchoscopy was normal. Esophagoscopy revealed varices limited to the upper third of the esophagus.

Roentgen Findings. The initial chest roentgenogram (Fig. 1) showed linear infiltration in both lungs, more prominent in the right upper lobe. A small but well defined shadow projected to the left and blended with the outline of the upper descending aorta. This represented a dilated accessory hemi-azygos vein. The significance of this finding as a sign suggesting superior vena caval obstruction has been described by Steinberg.[12]

An esophagram demonstrated varices limited to the upper third of the esophagus (Fig. 2).

Serial venograms, using 25 cc. of 50 per cent hypaque injected through an antecubital vein in each arm, clearly demonstrated the site of obstruction of the superior vena cava to be above the entry of the azygos vein (Fig. 3, *A*, *B*, *C* and *D*). The smooth tapered character of the occlusion suggested an inflammatory

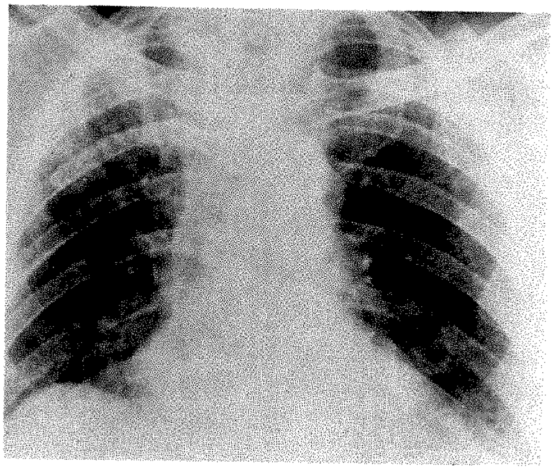

FIG. 1. Case I. Linear infiltration principally in the right upper lobe. Note the small shadow below the aortic arch which blends with the descending aorta. This was shown on venography to be a dilated accessory hemi-azygos vein, and is a sign of superior vena caval obstruction.

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stricture of the vessel. The left internal mammary vein and the lateral thoracic vein were dilated, as were superficial collaterals on the chest wall. The accessory hemi-azygos vein was enlarged. An unusual network of tortuous veins was noted in the mediastinum in both the anteroposterior and lateral views at the same level as the eosphageal varices. The direction of venous flow in these mediastinal collaterals as well as in the superficial veins draining the upper extremity was *downward*, blood eventually reaching the right atrium via the unobstructed azygos vein.

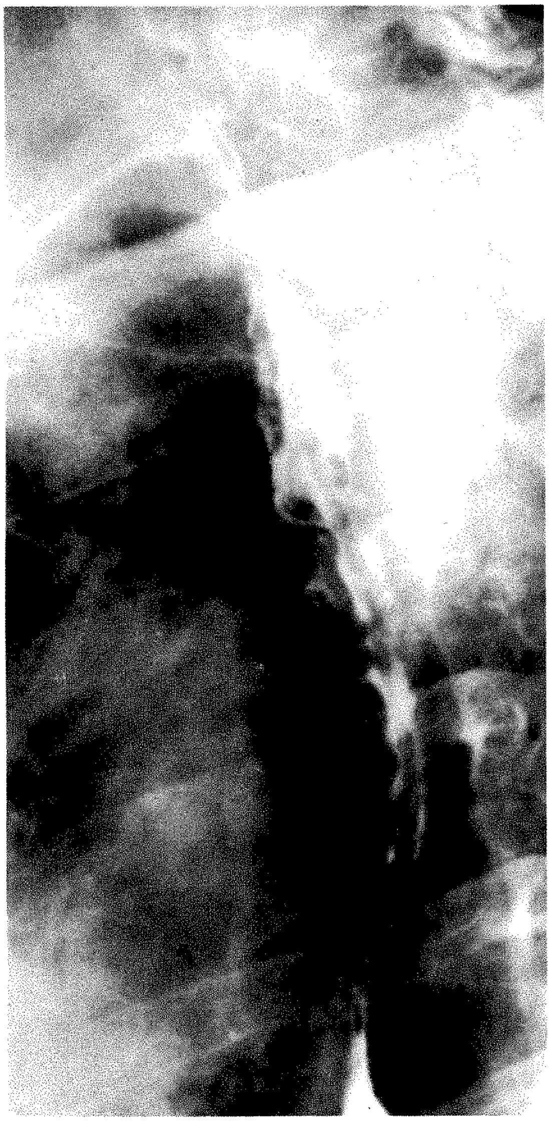

Fig. 2. Case I. Spot roentgenogram showing varices of upper esophagus.

Because of the patient's age, cerebrovascular disease, and lack of clinical disability referable to the caval obstruction, mediastinal exploration was not deemed advisable. He is still alive 2 years later.

Case II. W.D., a 35 year old Negro male, consulted a physician because of puffy eyelids and slight exertional dyspnea for the preceding 4 months. In addition, swelling of his neck requiring a larger size collar was noted. His only previous illness was a 3 month hospitalization in a tuberculosis sanitarium in 1951 because of an abnormal chest roentgenogram, but active tuberculosis could not be proved bacteriologically. He was discharged and followed at yearly intervals. He did not drink alcoholic beverages and denied gastrointestinal symptoms.

Physical Examination. There was edema of the face and eyelids, and distended neck veins. Dilated superficial veins which filled from above were present on the anterior chest wall. The liver and spleen were not enlarged.

The tuberculin skin test (second strength PPD) was positive; histoplasmin was negative.

Laboratory Data. Routine blood studies and urinalysis were normal. Liver function studies including serum bilirubin, serum proteins, and A/G ratio, alkaline phosphatase, thymol turbidity, and cephalin flocculation were normal. Bromsulphalein retention was 25 per cent at 45 minutes when performed via an antecubital vein, but normal when a foot vein was used. Gastric washings for acid fast bacilli were negative on smear and culture on two occasions.

A scalene lymph node biopsy (January 30, 1961) revealed reactive hyperplasia of 2 peasized lymph nodes.

Roentgen Findings. The admission chest roentgenogram (Fig. 4) showed linear infiltration in the right lung with slight loss of volume. The superior mediastinum was widened. Compared to previous roentgenograms in 1951 and 1956, there had been no change in the lung findings but the superior mediastinum had become wider.

An esophagram (Fig. 5) demonstrated the unexpected finding of huge varices involving almost the entire esophagus.

A serial venogram, made via the right antecubital vein using 50 cc. of 50 per cent hypaque (Fig. 6) disclosed collateral circulation in superficial veins over the chest wall flowing in a downward direction. The precise point of ob-

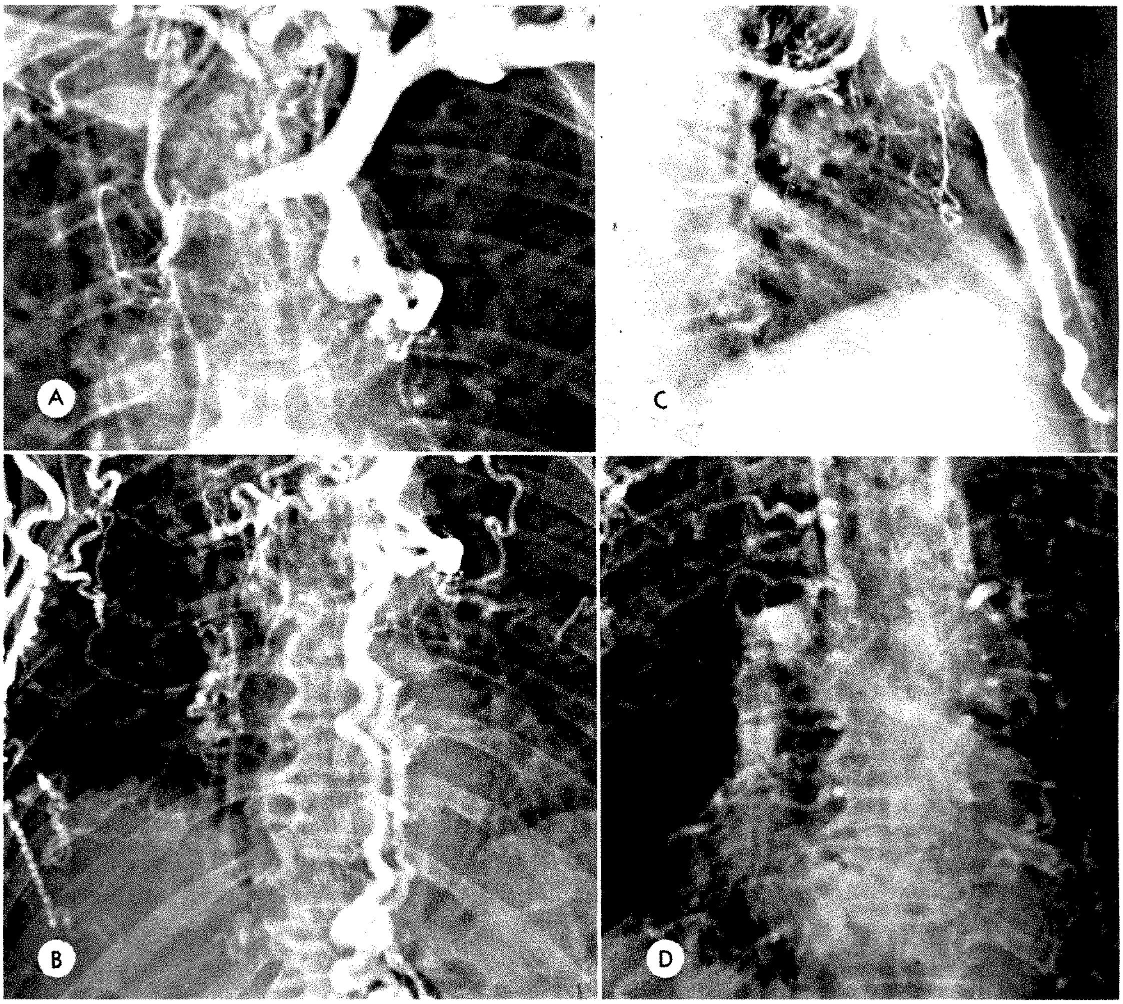

FIG. 3. Case I. Serial venacavagrams. (*A*) Three seconds. The obstructed vena cava ends as a tapered cone above entry of the azygos vein. Note beginning downward flow in mediastinal collaterals below point of obstruction as well as collaterals in midline communicating with dilated accessory hemi-azygos vein. The internal mammary vein is also dilated. (*B*) Five seconds. Further filling of mediastinal collaterals from accessory hemi-azygos vein and also below point of caval obstruction. These collaterals are at the level of the esophageal varices. Extensive downward collateral flow in the internal mammary, lateral thoracic veins as well as in chest wall is also present. (*C*) Five seconds. Lateral view (separate injection). The dilated accessory hemi-azygos vein arches down and posteriorly and feeds a network of collaterals in the mid-mediastinum precisely at the level of the varices, and also in the anterior mediastinum. (*D*) Fifteen seconds. Collateral flow in the paravertebral and intercostal veins returns blood to the heart via the unobstructed azygos vein.

struction of the vena cava could not be demonstrated.

A serial intraosseous azygogram, using 10 cc. of 50 per cent hypaque injected into each ninth rib posteriorly, showed no filling of the azygos vein beyond its mid-portion, with reversal of flow into the paravertebral veins (Fig. 7).

These findings suggested obstruction of the azygos vein as well as of the superior vena cava.

The patient refused surgical exploration of the mediastinum. He was alive and had no serious disability 2 years later, but has not been seen in the past year.

CASE III. A 48 year old white male was found to have an abnormal chest roentgenogram on a routine pre-employment examination. He had no symptoms referable to the chest although he had consulted a physician 2 weeks previously because of slight swelling of the face and eyelids, worse in the morning and subsiding as the day progressed. He had smoked $1\frac{1}{2}$ to 2 packages of cigarettes daily for 25 years, and drank beer "occasionally." There had been no serious previous illness.

Bronchoscopy revealed a "furrowed" right main bronchus but was otherwise normal.

Skin tests for tuberculosis and fungi were normal.

Physical Examination. There was slight swelling of the periorbital tissues, face and neck in the supine position and slight neck vein distention. There were no dilated veins on the chest wall.

Laboratory Data. Hb 12.0 gm. per cent. Liver function tests were normal. Sputum samples and bronchial washings were negative for malignant cells and no acid-fast organisms were found on direct smear or culture.

Roentgen Findings. A posteroanterior chest roentgenogram (Fig. 8) showed linear infiltration in the right upper lobe and elevation of the short interlobar fissure. The superior mediastinum was widened on the right side. The right hilus was enlarged and a smoothly rounded density projected just above the hilus in the position of the azygos vein.

An esophagram (Fig. 9) revealed varices extending from the aortic arch to the diaphragm.

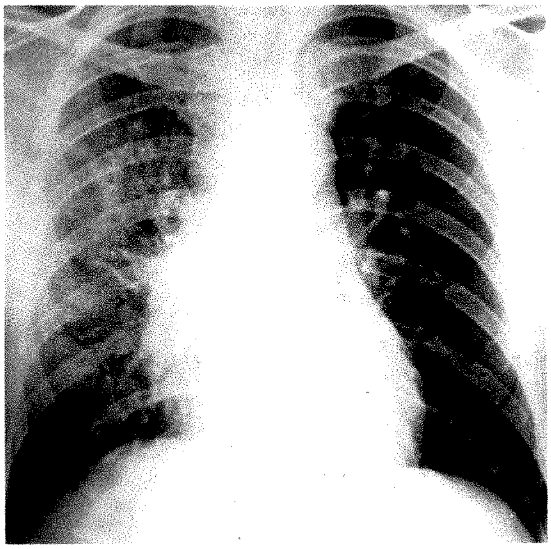

Fig. 4. Case ii. Admission chest roentgenogram. There is linear infiltration in the right lung, unchanged since 1951. The superior mediastinum is widened whereas it was normal in 1956.

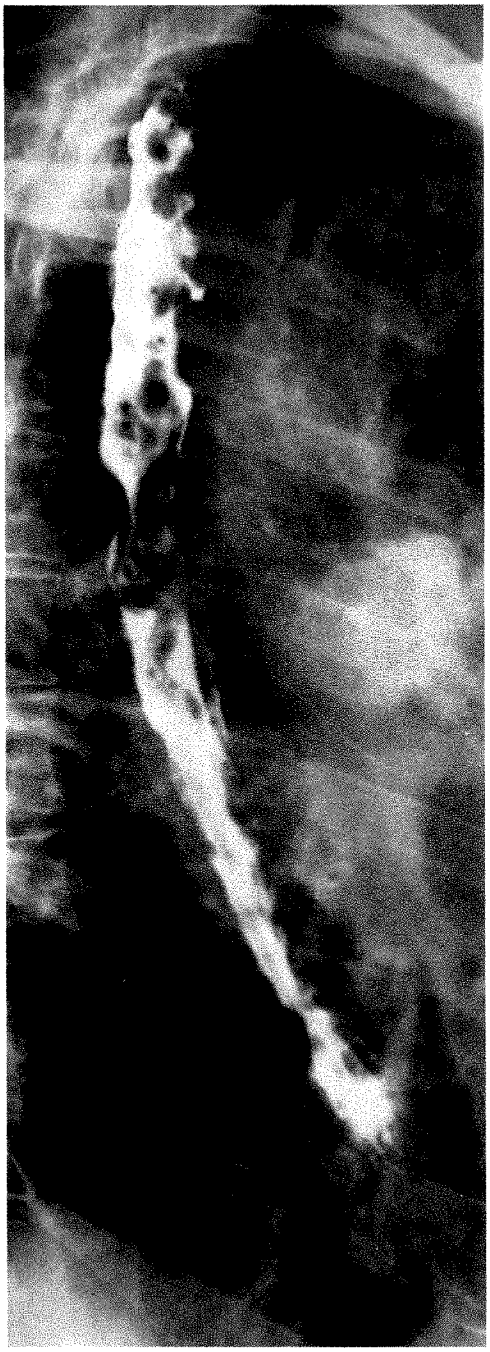

Fig. 5. Case ii. Esophagram showing large varices involving the entire esophagus.

On bronchography, the anterior segmental bronchus of the right upper lobe was obstructed, suggesting neoplasm (Fig. 10).

A superior venacavagram (Fig. 11) demon-

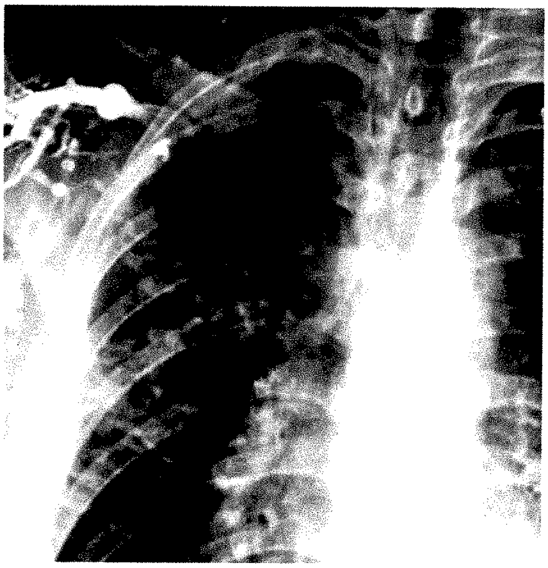

FIG. 6. Case II. Superior venacavagram. The point of obstruction could not be demonstrated due to high pressure in the vena cava. Collateral flow in lateral thoracic vein flowing downward is seen.

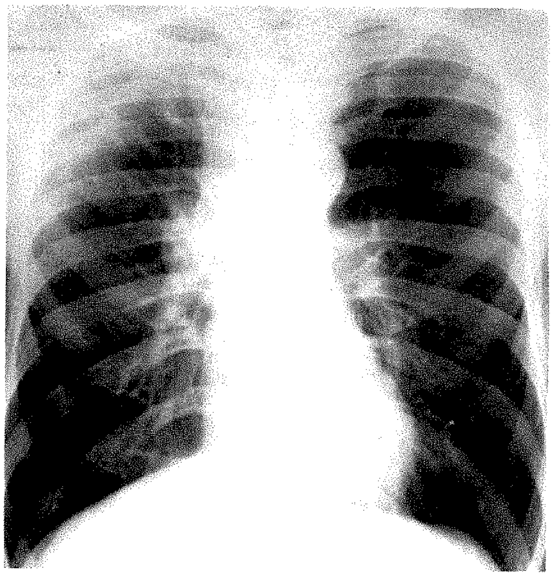

FIG. 8. Case III. There is linear infiltration in a slightly collapsed right upper lobe. The mediastinum is widened on the right side and the right hilus is enlarged. Note the smooth rounded density above the hilus which represents a dilated azygos vein.

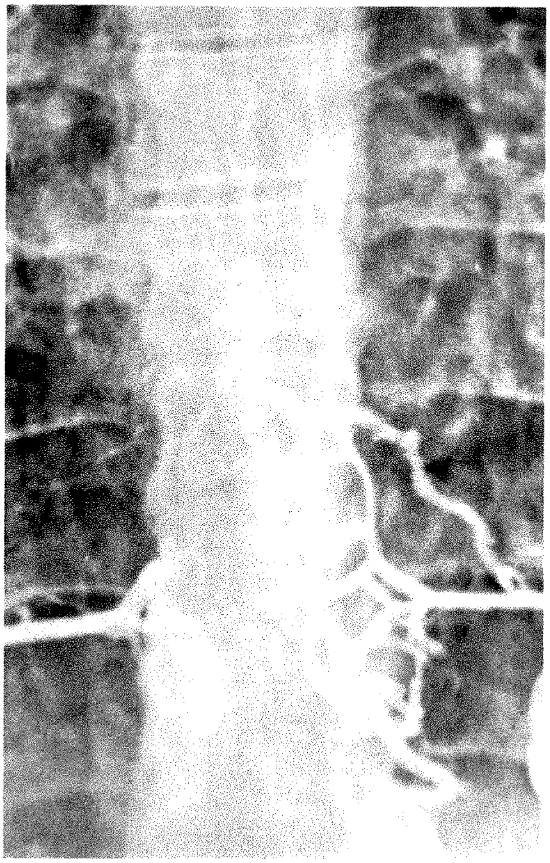

FIG. 7. Case II. Serial azygogram, 15 seconds. The proximal azygos vein could not be filled and there is reversed flow in the azygos and paravertebral systems.

strated partial stenosis of the vena cava below the entrance of the azygos vein. The azygos vein was dilated at its junction with the vena cava and the direction of venous flow was reversed; *i.e.*, downward. Similarly, the accessory hemi-azygos vein was dilated and the direction of flow was also downward.

Because of the history and roentgen findings, radiation therapy was administered to the mediastinum with symptomatic relief and prompt regression of the changes noted on the first chest roentgenogram. The patient is still under treatment.

CASE IV. J.W., a 54 year old Chinese male, had complained of a cough with blood-tinged sputum and tightness in the chest for 2 months. He had lost 15 pounds of weight during this period. Swelling of the face and neck, dizziness and temporary blurring of vision during paroxysms of coughing, hoarseness, and dyspnea progressing to orthopnea had developed in the 3 weeks before admission to the hospital. He had smoked 2 packages of cigarettes a day for many years, but denied using alcoholic beverages. There was no history of previous illness.

Physical Examination. The face and neck were swollen. Distended neck veins and superficial chest wall veins filled from above. The liver and spleen were not palpable.

Venous pressure in the upper extremities (antecubital veins of both arms) was 380–400 mm. of water as contrasted to 80 mm. of water in the right femoral vein. Arm to tongue circu-

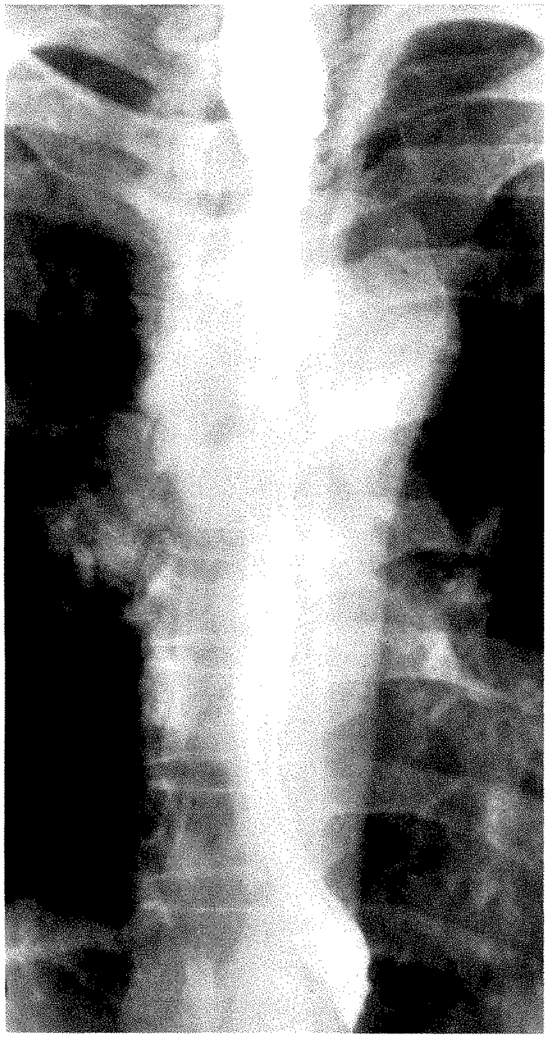

Fig. 9. Case III. Esophagram showing varices involving esophagus from the level of the aortic arch to the diaphragm.

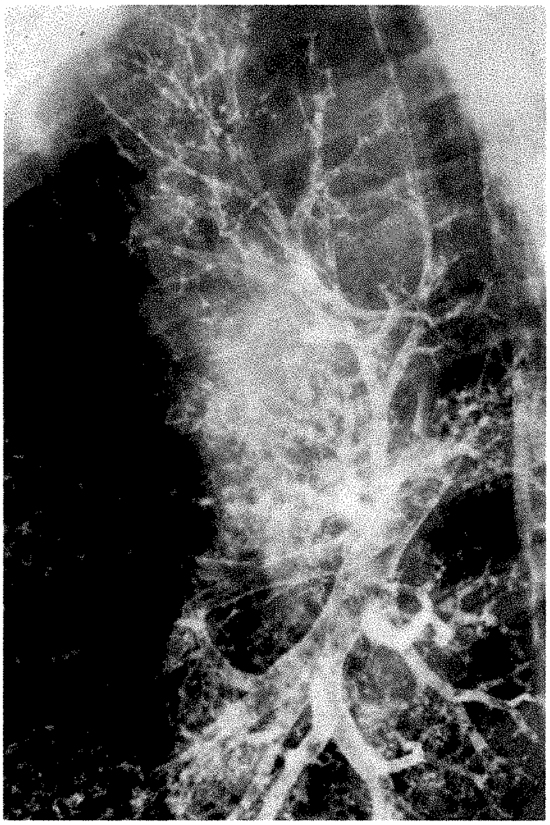

Fig. 10. Case III. Right bronchogram. There is loss of volume of the upper lobe. The anterior segmental bronchus is occluded suggesting neoplasm.

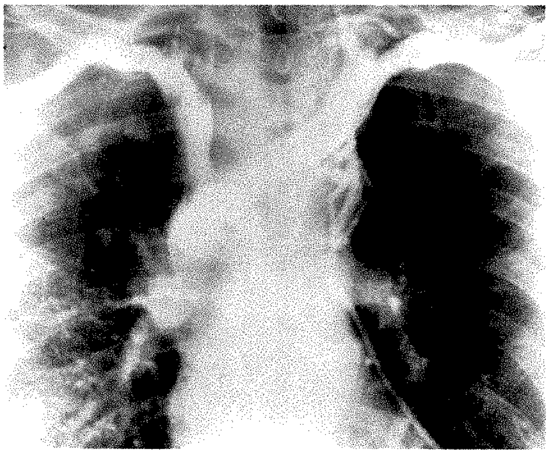

Fig. 11. Case III. Superior venacavagram. The vena cava is partially obstructed below the azygos vein, and flow in the azygos vein is retrograde. Note the collateral vessels in the mediastinum (projected over spine) in which blood flow is downward.

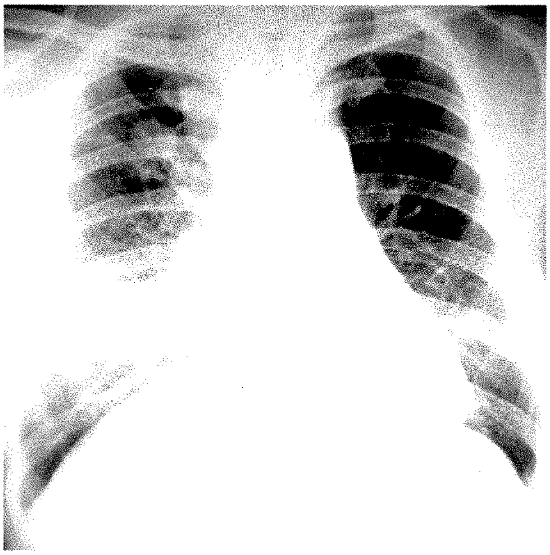

Fig. 12. Case iv. There is a 3 cm. nodular lesion in the right upper lobe. (No calcification was present on laminagrams.) The right superior mediastinum is widened. The convex shadow projecting into the right lung was thought to be the dilated or displaced superior vena cava.

lation time (decholin) in the upper extremities was prolonged to 45 seconds as compared with a normal time of 16 seconds in the lower extremity.

Skin tests for tuberculosis, histoplasmosis, blastomycosis and coccidioidomycosis were negative.

Bronchoscopy was normal, but the left vocal cord was found to be paralyzed.

Right scalene lymph node biopsy was normal.

Laboratory Data. Hb 11.0 gm. per cent. Liver function tests were normal. No malignant cells were found in multiple sputum samples, nor could acid-fast organisms be identified on smear or culture.

Roentgen Findings. A posteroanterior chest roentgenogram (Fig. 12) revealed a 3 cm. nodular lesion in the right upper lobe in which no calcification was demonstrated on laminagraphy. The superior mediastinum was widened and a smooth convex shadow, believed to represent a dilated superior vena cava, projected from the mediastinum into the right lung.

An esophagram (Fig. 13) showed varices involving the upper half of the esophagus.

Superior venacavagrams (Fig. 14, *A* and *B*; 15, *A* and *B*; and 16) using the technique described previously showed evidence of high

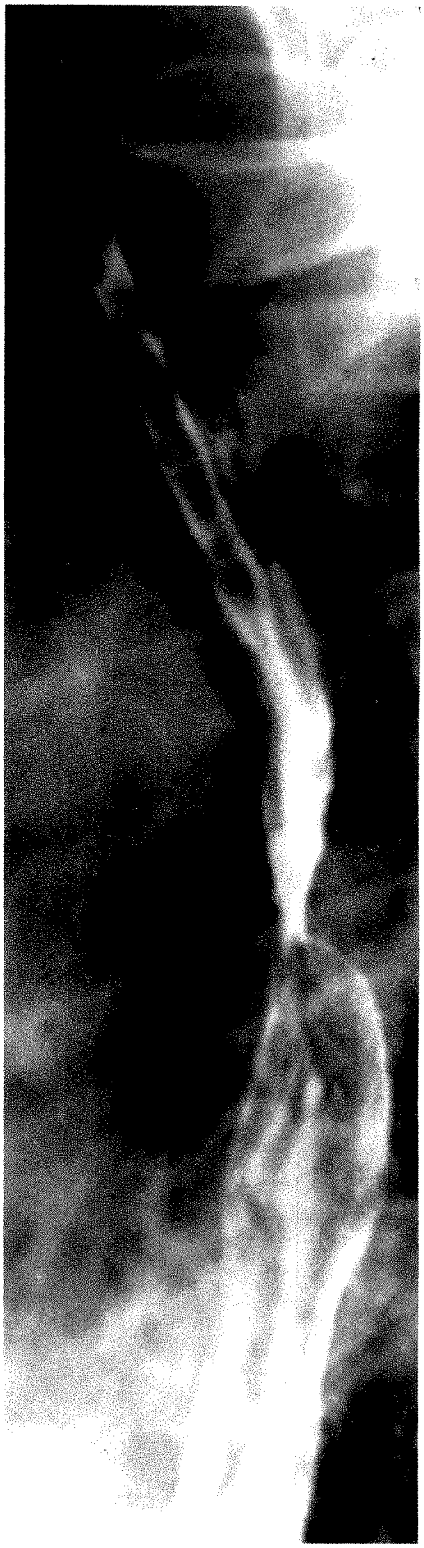

Fig. 13. Case iv. Spot roentgenogram showing varices localized to the upper half of the esophagus.

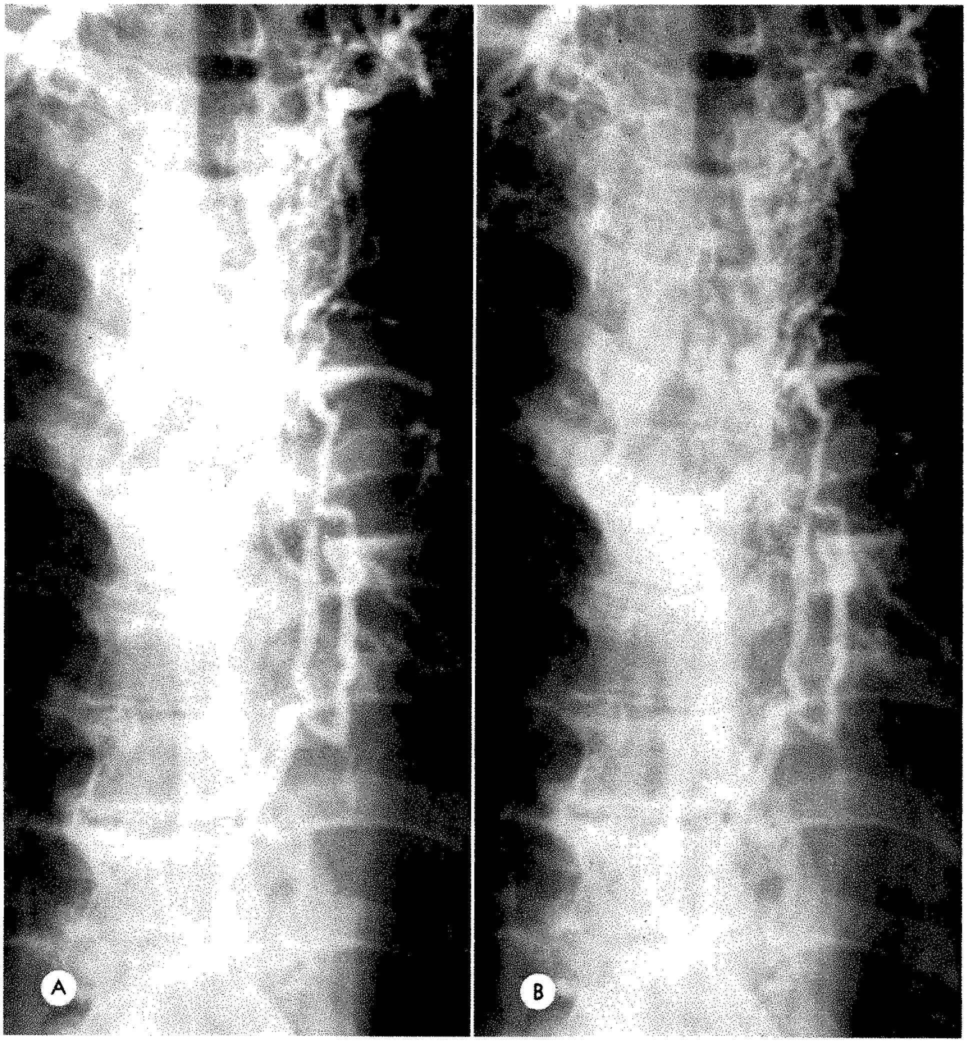

Fig. 14. Case iv. (*A* and *B*) Superior venacavagrams, 5 and 8 seconds. The level of vena caval obstruction is not identified because of high pressure in the system. Note downward flow in mediastinal collaterals and accessory hemi-azygos vein with opacification of lower azygos vein (to right of spine) to level of T-8. There was no opacification of the azygos vein at junction with vena cava or the right atrium on delayed studies.

pressure in the vena cava although the precise point of obstruction was not demonstrated. Extensive mediastinal collateral circulation, flowing *downward*, was noted in both the anteroposterior and lateral views. Some of these veins were intimately related to the upper half of the esophagus at the level of the varices.

The symptoms of superior vena caval obstruction promptly regressed after one course of intravenous nitrogen mustard and radiation therapy to the mediastinum, but shortly thereafter malignant cells were recovered from a right pleural effusion, and osteolytic metastatic lesions developed in the lumbar spine and sacrum.

DISCUSSION

There are normally extensive anastomoses between the caval, portal, azygos, and vertebral venous systems allowing multiple routes for the return of blood to the heart (Fig. 17). The esophageal veins normally communicate with both the portal and azygos systems. The veins of the lower esophagus drain into the portal vein

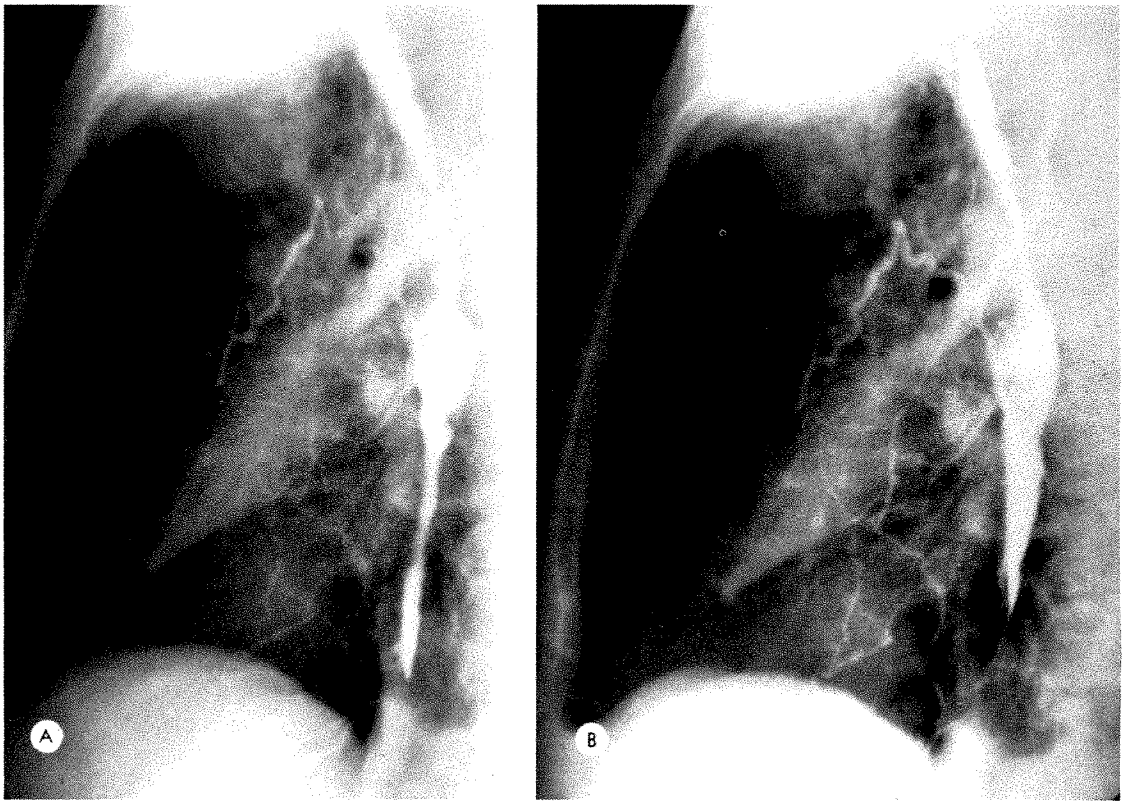

Fig. 15. Case iv. Lateral view with barium in esophagus. (*A*) 4 seconds; (*B*) 8 seconds. Note downward flow in accessory hemi-azygos vein communicating with collateral veins in mediastinum and also adjacent to the upper half of the esophagus at level of varices.

through the left gastric vein and also anastomose with the azygos system; the mid-esophagus is drained by the azygos and hemi-azygos veins.[6]

Obstruction of the superior vena cava and portal hypertension may cause esophageal varices, but the direction of flow in the varices differs in the two conditions. In portal hypertension, one of the available collateral routes is *upward* through the left gastric and esophageal veins, blood reaching the heart via the azygos and superior vena cava. With obstruction of the superior vena cava, collateral channels in the mediastinum may enlarge, communicate with the esophageal veins and result in varices, but the direction of flow in these varices is *downward*. Blood returns to the right heart through either the azygos or the inferior vena cava, depending on the level of occlusion of the superior vena cava (Fig. 18, *A* and *B*).

Carlson[3] ligated the superior vena cava in dogs and showed that the azygos vein and its tributaries formed the chief means of returning blood to the heart from the head and upper extremities when the vena cava was ligated above the azygos. The superficial and deep abdominal vessels, together with the vertebral plexus, were the main collateral routes when both the vena cava and the azygos vein were occluded. As the azygos vein was blocked, all of the blood returned to the heart via the inferior vena cava. He also noted well developed mediastinal collateral veins regardless of the level of obstruction, although he did not describe esophageal varices.

Duchek,[4] in a comprehensive description of the collateral circulation in superior vena caval obstruction, observed upper esophageal veins communicating with the accessory hemi-azygos and anastomoses between lower esophageal veins, the azygos vein, and the portal system. He pointed out that though the superior vena cava and the azygos vein were obstructed, the inferior portion of the azygos system may be patent and allow collateral circulation from the vertebral plexus and intercostal veins to reach the inferior vena cava. Thus reversal of flow in the azygos system could develop and possible routes for blood from the head and upper extremities to return to the heart would be: vertebral plexus, intercostal veins, patent lower azygos and esophageal veins to the left gastric, portal vein, and inferior vena cava (Fig. 18*B*).

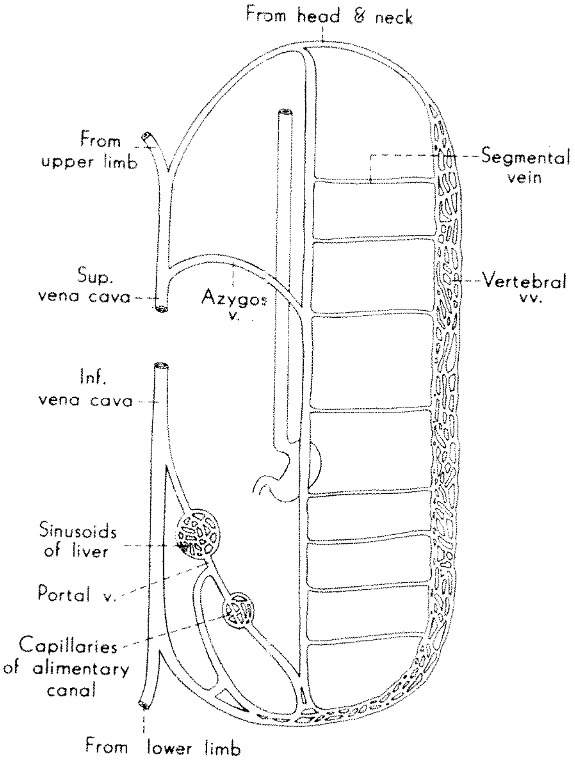

FIG. 17. Case IV. Schematic diagram of venous circulation. The azygos and vertebral systems, in which blood flow is normally reversible, bypass the caval systems. (Reproduced by permission from W. B. Saunders Company.[6])

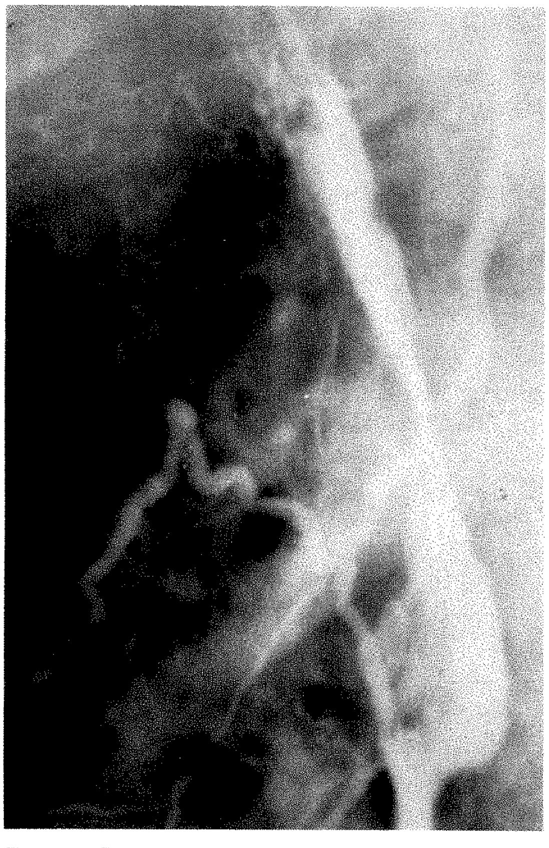

FIG. 16. Case IV. Magnified view of Figure 15. Note the network of collateral vessels adjacent to the esophagus at the level of the varices.

Varices may develop on the basis of vena caval obstruction alone, contrary to the explanation offered by Snodgrass and Mellinkoff[11] that the azygos vein as well as the superior vena cava must be occluded.

The level of vena caval obstruction was proximal to the azygos vein in one patient (Case I) and a partial obstruction below the azygos vein was present in another (Case III). In the former, one of the routes of blood from the head and upper extremities to return to the heart was through the unobstructed azygos vein via mediastinal collaterals. The point of obstruction could not be determined in the other 2 patients, although extensive collateral circulation was demonstrated in both. As Bergstrand[2] has pointed out, failure to show the point of obstruction may be due to high pressure in the obstructed vessel relative to the

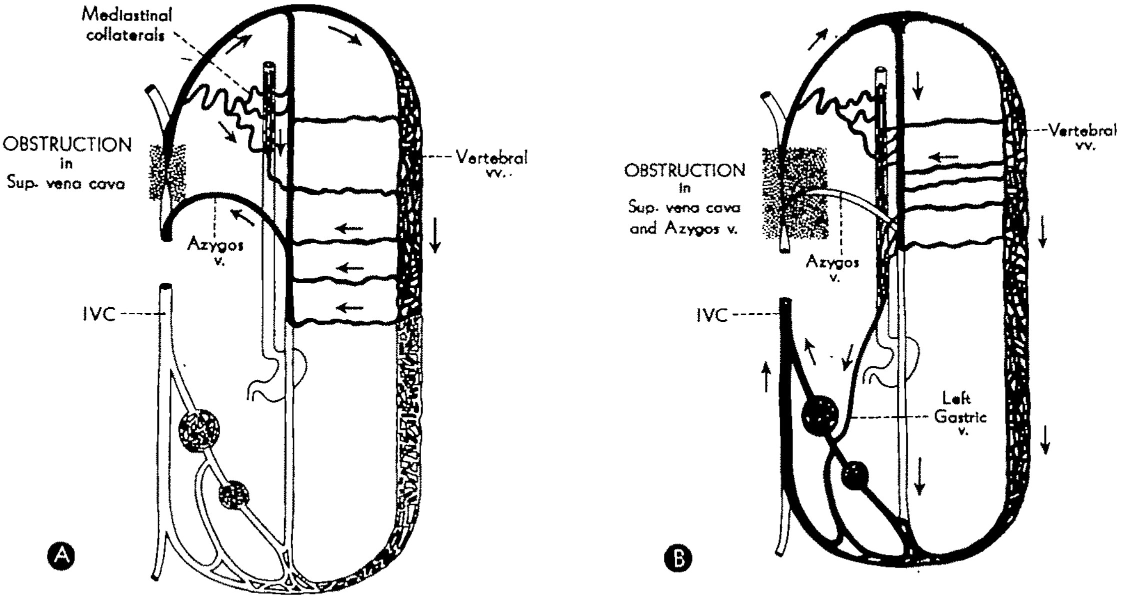

FIG. 18. Collateral circulation producing esophageal varices in superior vena caval obstruction. (*A*) Obstruction above azygos vein. One route is downward in mediastinal collaterals and reversed flow in vertebral and intercostal veins both communicating with esophageal veins, accessory hemi-azygos vein and azygos systems. Blood returns to heart via azygos vein. (*B*) Obstruction involving azygos vein. Blood from head and upper extremities can reach inferior vena cava through a mediastinal route via the accessory hemi-azygos vein, esophageal veins, patent lower azygos vein, left coronary and portal veins.

pressure of injection resulting in run off in collateral vessels.

In one patient (Case II) with extensive varices presumed to result from mediastinal fibrosis, neither the point of vena caval obstruction nor the mediastinal collateral circulation could be demonstrated, but the proximal azygos vein did not fill on azygography and flow was reversed in the patent distal azygos and paravertebral veins. Although retrograde flow in the azygos system may occur normally, as Schwartz *et al.*[10] have observed, and is not significant unless accompanied by evidence of obstruction, in this case the findings suggested obstruction of the azygos vein as well as of the superior vena cava. The postulated collateral circulation in the mediastinum which produced esophageal varices was downward as Duchek postulated.

In 3 of the 4 patients, downward flow of blood in mediastinal collaterals was noted on anteroposterior and lateral venograms, and a network of collateral vessels was clearly shown at the precise level of the varices in 2 of these patients (Case I and IV).

CLINICAL CONSIDERATIONS

Including the 4 cases reported here, there are at present 20 known examples of esophageal varices secondary to superior vena caval obstruction (Table I). In addition, Weinberg[15] described 5 cases, found at autopsy, of varices associated with mediastinal metastatic bronchogenic carcinoma, although he did not state whether clinical vena caval obstruction had been present during life, and Sundermann and Kaemmerer[13] reported 2 examples of localized upper esophageal varices after resection of a retrosternal thyroid gland without vena caval obstruction.

The entity is probably not as uncommon as it would appear, as 16 of the 20 known cases have been reported in the past 2 years. Careful barium studies of the esophagus, specifically searching for varices,

TABLE I

SUMMARY OF REPORTED CASES
ESOPHAGEAL VARICES SECONDARY TO SUPERIOR VENA CAVAL OBSTRUCTION

Authors	No. of Cases	Etiology	Location of Varices	Bleeding
Templeton[14] 1944	1	Undetermined	Lower esophagus	No
Garrett and Gall[7] 1953	1	Hodgkin's disease	Not stated	Not stated
Kaplan[8*] 1941	2	Carcinoma	Not stated	Not stated
Snodgrass and Mellinkoff[11] 1961	1	Mediastinal fibrosis	Upper third	Yes
Mikkelsen[9] 1963	8	Mediastinal fibrosis (1)	Entire esophagus	No
		Bronchogenic carcinoma (5)	Upper half	No
		Adenocarcinoma of unknown origin (1)	Upper half	No
		Following neck dissection for thyroid carcinoma (1)	Upper half	No
Felson[5]	3	Mediastinal fibrosis (1) Bronchogenic carcinoma (2)	Entire esophagus Upper Third	No
Present Series	4	Mediastinal fibrosis (2)	Upper third Entire esophagus	
		Bronchogenic carcinoma (2)	Upper half Entire esophagus	No

* Reported 2 cases in German literature.

in all patients with superior vena caval obstruction may result in the discovery of additional cases.

Metastatic bronchogenic carcinoma and mediastinal fibrosis were the two most important causes of the vena caval obstruction in the reported cases. Although there is no histologic proof of etiology in our 4 patients, clinical evidence favors mediastinal metastatic carcinoma, primary in the lung, in 2 and fibrous mediastinitis, either histoplasmosis or idiopathic, in 2.

The prognosis is apparently related more to the underlying cause of vena caval obstruction than to the presence of varices. In mediastinal fibrosis, the symptoms of vena caval obstruction tend to regress as collaterals develop and the disease is self-limited,[1] while in carcinoma the prognosis is almost uniformly fatal.

If varices develop as part of the collateral circulation, the threat of bleeding exists. None of our 4 patients bled, and 2 with presumed mediastinal fibrosis have been followed for 2 years. One has had a history of vena caval obstruction for 7 years; neither has developed significant clinical disability. A case of profuse hemorrhage from varices associated with vena caval obstruction due to mediastinal fibrosis has been reported,[11] and there is no reason to assume that these varices may not bleed.

SUMMARY

1. Four patients without clinical evidence of liver disease or portal hypertension who

developed esophageal varices secondary to obstruction of the superior vena cava are described.

2. The routes of collateral circulation that may develop following superior vena caval obstruction are discussed with emphasis on the mediastinal collaterals.

3. Varices secondary to superior vena caval obstruction may be limited to the upper third or involve the entire esophagus, and are the result of downward flow of blood from the head and neck in mediastinal collaterals. In 2 patients, downward flow in extensive mediastinal collaterals was clearly demonstrated on venograms to be precisely at the level of the varices.

4. Mediastinal fibrosis and bronchogenic carcinoma have been the two most important causes of this entity, with the prognosis depending upon the basic cause of vena caval obstruction. Bleeding from these varices has been reported in only 1 of 20 patients.

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We wish to express our appreciation to Dr. Eugene Harkaway, Radiologist, Herman Kiefer Hospital, Detroit, Michigan, for his permission to use Case III.

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HERNIATION OF THE TRANSVERSE COLON THROUGH THE FORAMEN OF MORGAGNI SIMULATING LEFT VENTRICULAR HYPERTROPHY*

REPORT OF A CASE

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HERNIATIONS through the foramen of Morgagni are uncommon, but are true internal diaphragmatic hernias that have a peritoneal hernial sac. The hernia passes through a retrosternal congenital defect in the anterior medial insertion of the diaphragm, termed the foramen of Morgagni or Larrey's fissure. The descriptive designation, "subcostal sternal diaphragmatic hernia," is more informative than the eponym and is coming into common use. A difference of opinion exists as to whether these hernias are congenital in origin or represent acquired defects.

Subcostal sternal herniations are the least common type of diaphragmatic hernias.[4] In a series of 3,395 hernioplasties, only 40, or 1.2 per cent, were diaphragmatic.[1] Of these, a small, unreported number was through the foramen of Morgagni. In Harrington's series of diaphragmatic hernias, 8 of 430 were subcostal sternal.[5] In the majority of reported cases, the diaphragmatic defect was on the right side of the sternum with the hernial sac presenting in the lower right hemithorax. This right-sided location is often associated with nonrotation of the right side of the colon. Of the 8 subcostal sternal hernias in Harrington's series, 6 were on the right and 1 was bilateral. Saltzstein *et al.*[6] presented the largest series of subcostal sternal diaphragmatic hernias, collecting 44 cases from the literature and adding 1 additional case of their own. Of the total 45 cases, 32 were on the right, 10 were bilateral, and only 3, or 8 per cent, presented solely on the left.

The contents of the hernial sac vary widely; stomach, omentum, and colon, singly or in any combination, may be found within the sac. Patients may be in acute distress or completely asymptomatic, as in the case reported below. Symptoms experienced by the patient are usually those most often associated with abnormality in the organ found within the hernial sac. Complete and partial intestinal obstruction, respiratory distress, dyspnea, cough, obstipation and constipation, and chest and/or abdominal pain are among the varied presenting complaints.

In the following case, the diagnosis was first suspected on a routine 70 mm. photofluorogram of the chest. Follow-up roentgenographic examination established the diagnosis.

REPORT OF A CASE

The patient was a 20 year old while male. As part of a routine physical examination prior to separation from the United States Army, an apparent enlargement of the left heart border was observed on a 70 mm. photofluorogram, but closer inspection suggested radiolucent defects within the enlarged cardiac silhouette (Fig. 1). Chest roentgenograms were then made and the presence of radiolucent pockets overlying the left heart border and in close association with the cardiac silhouette was confirmed (Fig. 2 and 3). A tentative diagnosis of hernia through the foramen of Morgagni was made.

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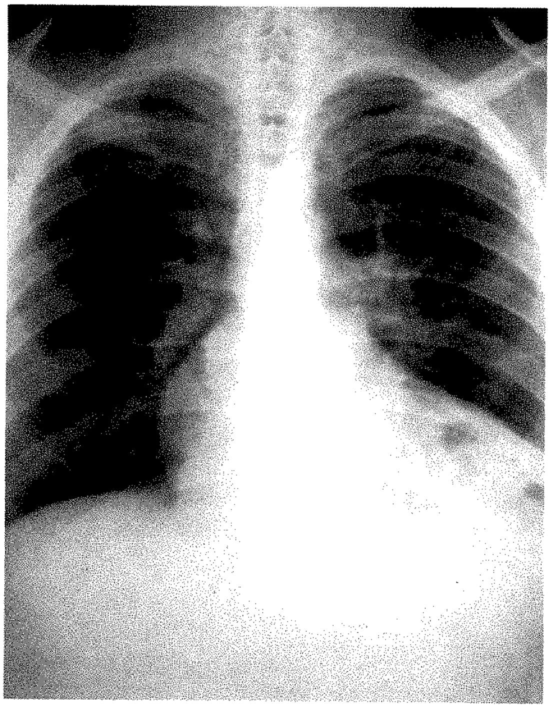

FIG. 1. Apparent left ventricular hypertrophy is noted on a posteroanterior chest roentgenogram which is similar to that observed on the original 70 mm. photofluorogram. Close inspection reveals several radiolucent defects within the apparently enlarged cardiac silhouette. The presumptive diagnosis of herniation through the foramen of Morgagni was made from this examination.

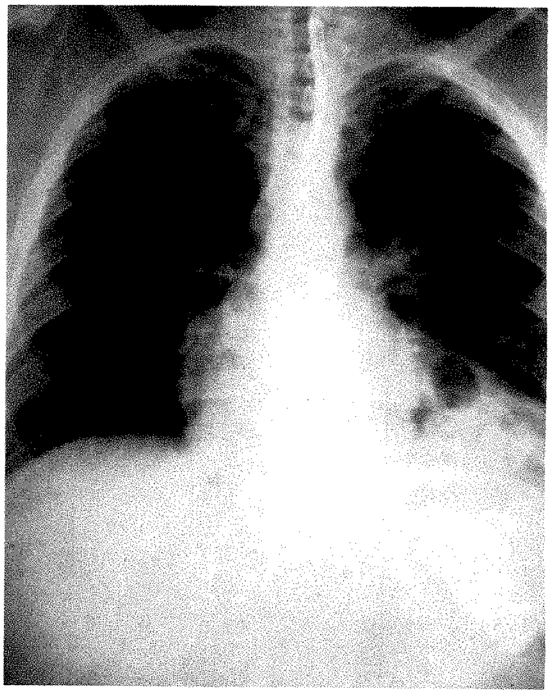

FIG. 2. On a repeat posteroanterior roentgenogram of the chest with the esophagus filled with barium, enlargement and apparent change in contour of the gas-filled radiolucent areas are noted. There is no definite evidence of cardiomegaly and the peripheral lung fields appear clear.

The patient had been in excellent health and was completely asymptomatic. Review of his induction physical examination revealed a report of enlargement of the left cardiac shadow on his chest roentgenogram. On several occasions, this was interpreted as left ventricular hypertrophy. His past history was noncontributory except for the fact that he had played semiprofessional football prior to his entry into the Army. He had had no abdominal or thoracic operations and no significant injuries.

Physical Examination. Increased pericardial dullness was present to percussion in the left anterior axillary line in the fifth intercostal space and bowel sounds were heard on auscultation over the cardiac area. The remainder of the examination was negative.

Laboratory Data. Blood urea nitrogen 10.6 mg. per cent; fasting blood sugar 96 mg. per cent; normal urinalysis; prothrombin time 88 per cent; white blood cell count 6,350 with normal differential; hematocrit 49 per cent; hemo-

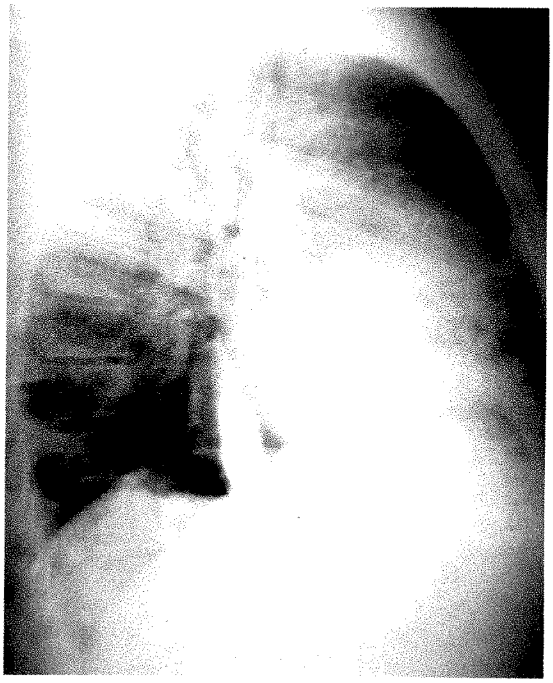

FIG. 3. The gas-filled bowel is seen anteriorly and subcostosternally in the lateral roentgenogram.

globin 16.3 gm. per cent; electrocardiogram was within normal limits.

Roentgen Findings. Gastrointestinal study of the esophagus, stomach and small intestine was negative. During barium enema examination, herniation of approximately 30 cm. of the splenic flexure and distal portion of the transverse colon through the foramen of Morgagni into the left lower hemithroax was observed (Fig. 4 and 5). The hernial sac and the pericardium appeared to be contiguous and the possibility that the hernia might enter the pericardial sac was considered.

Hospital Course. On February 14, 1963, a transthoracic repair of the hernia was performed. A congenital absence of approximately 5 cm. of the most medial portion of the left hemidiaphragm at its attachment to the seventh rib and the xiphoid was noted. There was considerable scarring of the large hernial sac which measured 25 cm. in length and was adherent to, but did not enter, the pericardium. The patient had an uneventful postoperative course and was discharged on February 25, 1963 in excellent health. In a postoperative chest roentgenogram, a normal cardiac silhouette and no evidence of the mass previously present were noted (Fig. 6).

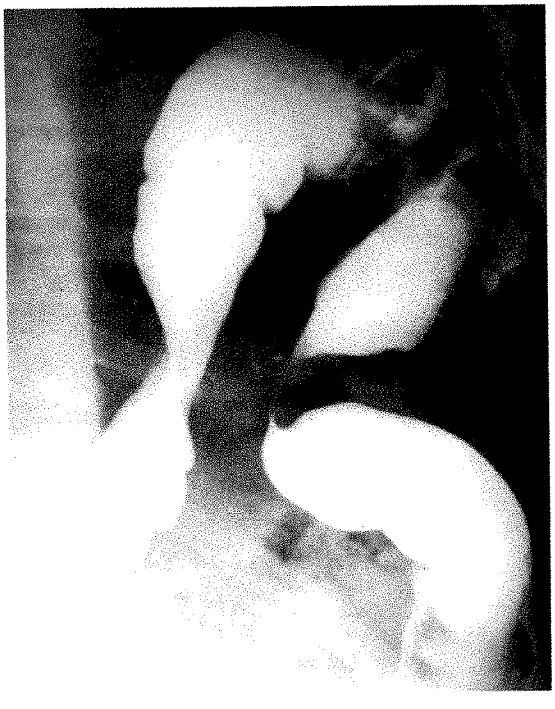

FIG. 4. A large segment of transverse colon is seen within the left hemithorax in the barium enema study. The apparent narrowing of the colon is at the level of the left hemidiaphragm where the bowel transcends the diaphragmatic defect through the foramen of Morgagni.

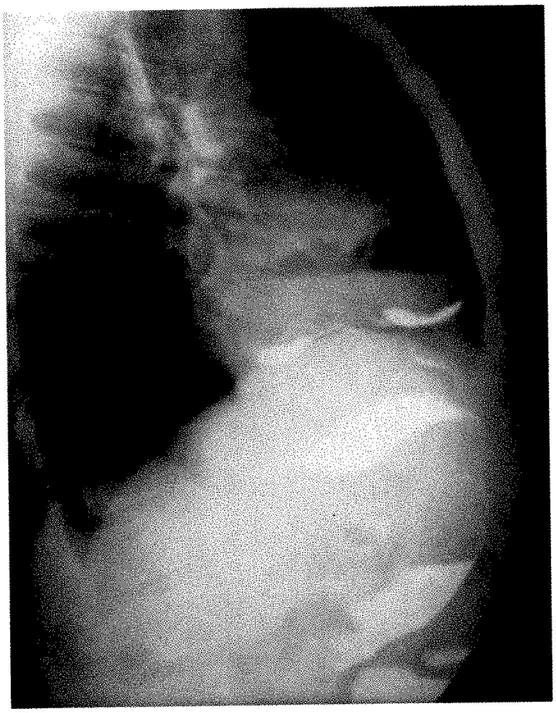

FIG. 5. An upright lateral roentgenogram of the chest shows the bowel with air-fluid levels and barium anteriorly.

DISCUSSION

The diagnosis of subcostal sternal herniation may be suspected on survey roentgenograms of the chest when gas-filled pockets are seen within the chest. In the case presented, the patient was reported to have left ventricular hypertrophy. This provisional diagnosis was made at another hospital on the basis of apparent enlargement of the cardiac silhouette on the chest roentgenogram. In the photofluorogram made at this institution, air was noted to lie within the apparently enlarged cardiac silhouette. In the lateral projection of the chest (Fig. 2), the gas pattern was seen to be anterior and subcostal sternal in location. The posteroanterior and lateral roentgenograms were sufficient to permit the

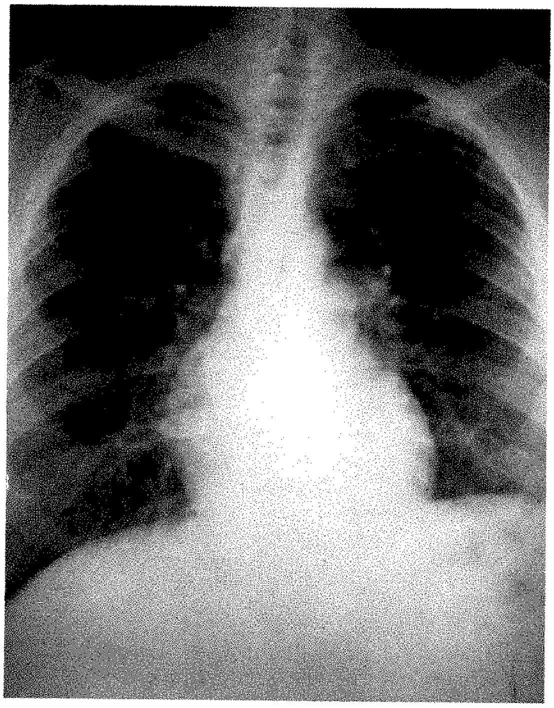

Fig. 6. A normal cardiac silhouette is seen in the postoperative posteroanterior roentgenogram of the chest. Slight elevation of the left hemidiaphragm is noted.

presumptive diagnosis of subcostal sternal herniation.

The diagnosis is usually established by routine barium studies. The anatomic structures within the hernial sac may be observed on upper gastrointestinal or barium enema examination. Because various combinations of abdominal viscera may be within the hernial sac, both examinations should be performed. The gas-filled loops of bowel may suggest a small bowel pattern or haustral markings of the colon; however, gas may be obscured by large amounts of omentum within the sac.[2] Occasionally, a pneumoperitoneum may be required to reveal the anatomic structures within the hernia. In the case presented, the upper gastrointestinal and small bowel studies were normal and the barium enema study revealed the hernia to contain only colon. The barium enema examination confirmed the diagnosis made from the roentgenograms of the chest.

SUMMARY

Subcostal sternal hernias (foramen of Morgagni hernias) are unusual. They are even more uncommon when they present solely on the left. The patient reported was asymptomatic. On routine examination of the chest, the left cardiac border was apparently enlarged. Air-filled defects were seen within the cardiac silhouette. A large segment of transverse colon was seen within the left hemithorax on barium enema examination and the diagnosis of herniation through the foramen of Morgagni was established. The defect was repaired surgically and the roentgenographic appearance of the chest was normal postoperatively. This condition may be suspected when air is seen retrosternally in the anterior thorax.

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TRANSMIGRATION OR PROLAPSE OF ESOPHAGEAL MUCOSA*

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TRANSMIGRATION of the lower esophageal mucosa is receiving increasing attention in the medical literature. Palmer[8] pointed out that mysterious spontaneous migrations of mucosa occur in the region of the esophagogastric junction. He demonstrated this by fixing metal brain clips to the mucosa in this region during esophagoscopy and then observed their movements during barium studies of the esophagus under fluoroscopic control. He described the appearance of mobile mucosa propelling itself over the deeper tissues, and considered whether transhiatal gastric herniation might occur in normal people. Aldridge[1] reviewed the medical literature to 1962, and added 91 cases of transmigration of esophageal mucosa which he detected in 2,100 consecutive unselected upper gastrointestinal series. He used the term "transmigration" for the condition variously described as "gastroesophageal invagination," "invagination of the esophagus," "cardioesophageal intussusception," and "extrusion or prolapse of mucosa into the esophagus." He drew attention to the fact that relatively few cases had been reported in the literature. To date, no such cases have been reported in the Japanese literature.

The condition is mentioned only briefly in recent editions of some radiologic texts.[3,9] It is easily overlooked, primarily because of its transitory nature,[6] and is easily misinterpreted since its appearance is similar to that of hiatal hernia[5] and carcinoma or polypoid mass in the lower portion of the esophagus,[2,4] with which it can be confused. The condition is frequently associated with a sliding-type hiatal hernia, but hiatal hernia is apparently not a prerequisite for its presence, nor is mucosal prolapse an indication of the presence of hiatal hernia.[1]

Only one instance of strangulation or incarceration has been reported.[2] Mucosal prolapse is reportedly responsible for producing symptoms, though it seems less frequently responsible for them than hiatal hernia.[1] It is difficult when these coexist to determine the degree to which each condition is responsible for symptoms. When coexistent, the symptoms are probably increased in intensity.[6]

Mucosal prolapse is presumably best demonstrated when the patient is symptomatic.[6] The recumbent and Trendelenburg positions, with increase in intra-abdominal pressure, have been found most useful for detecting cases.[5,6,7] Klinefelter[6] has found that elevating the patient from the recumbent to the vertical position is advantageous in demonstrating the deformity, and that the prolapse is reduced during such procedure. Aldridge[1] states that the "vigor" under which the examination is performed is also a factor in producing the deformity. The appearance of the prolapse has been described by Klinefelter[6] as that of "jack in the pulpit," and Aldridge[1] has referred to an "arum lily" appearance, both descriptive of the intussusceptive type—the ring of barium in the likeness of the intussuscipiens being isolated from the main column of barium in the likeness of the intussusceptum. Wells[10] first described the retrograde type of transmigration and several other

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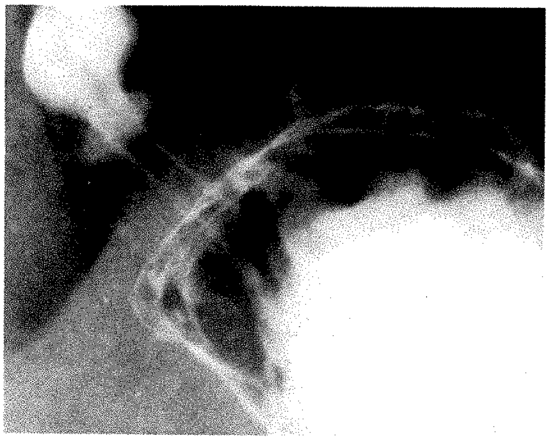

Fig. 1. Case i. Prograde transmigration.

cases have since been reported.[2,4,5] These produce a characteristic mushroom-like filling defect in the distal end of the esophagus causing momentary delay in the descent of the bolus due to a ball-valve action.

Of 5 cases detected here, the prolapsed type of deformities are exemplified in 4 cases, the retrograde type in 1 case.

REPORT OF CASES

Case i. MF # 869046. A 66 year old Japanese female with epigastric pain had no remarkable physical or laboratory findings. An upper gastrointestinal tract examination revealed a small sliding-type of hiatal hernia and an annular configuration of barium around the main esophageal barium column, indicating mucosal prolapse near the esophagogastric junction. A benign gastric ulcer was present on the lesser curvature of the stomach. There was transpyloric mucosal prolapse, but the stomach was otherwise not remarkable. The benign ulcer subsequently healed with relief of symptoms (Fig. 1).

Case ii. MF #264728. A 60 year old Japanese male with epigastric pain was suspected of having a gastric ulcer. The history and physical examination were not otherwise contributory; laboratory examination showed only slight eosinophilia. An upper gastrointestinal tract examination revealed a small hiatal hernia, 3.5 cm. long and 2 cm. wide. Moderate prolapse of the esophageal mucosa into the gastric segment was manifested by an annular configuration of barium around the main barium column in the esophagus near the esophagogastric junction. A gastric ulcer on the lesser curvature and transpyloric prolapse of the mucosa were present. This ulcer subsequently healed with minimal deformity and with resolution of symptoms (Fig. 2).

Case iii. MF #243202. A 78 year old Japanese female with anemia of unknown etiology complained of heartburn after meals, and a benign gastric ulcer or gastric carcinoma was suspected. An upper gastrointestinal series revealed a moderate size hiatal hernia, 5 cm. long and 3 cm. wide, and moderate mucosal prolapse at the esophagogastric junction, manifested by an annular configuration of barium about the main barium column in the esophagus. The stomach and duodenum were not remarkable, except for transpyloric prolapse of gastric mucosa. Whether the symptoms persisted is not known (Fig. 3).

Case iv. MF #476608. A 67 year old Japanese female had experienced epigastric pain unrelated to eating for several years. Physical examination revealed mild tenderness in the epigastrium on palpation. An upper gastrointestinal tract examination demonstrated a small hiatal hernia and mucosal prolapse in the midportion of the hernia with a "jack in the pulpit" appearance. The stomach and duodenum were not remarkable except for some transpyloric prolapse of the gastric mucosa (Fig. 4).

Case v. MF #466321. A 78 year old Japanese female had occasional heartburn. An upper gastrointestinal series 6 years previously was normal. Current examination of the upper gastrointestinal tract showed a sliding-type

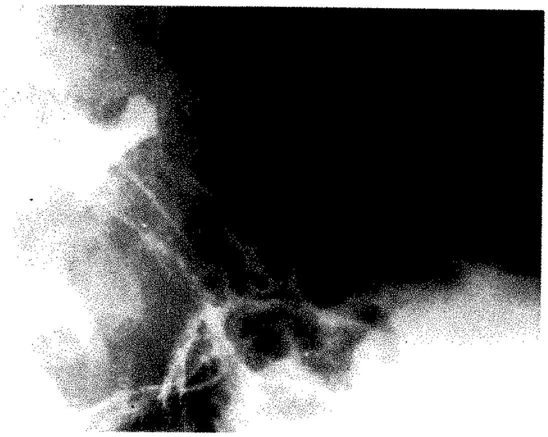

Fig. 2. Case ii. Prograde transmigration.

hiatal hernia with retrograde gastric mucosal prolapse into the distal end of the esophagus, causing indentation which was visualized by means of air contrast. Neither transpyloric mucosal prolapse nor other changes were evident. The patient was re-examined 2 years later. The gastrointestinal symptoms had subsided and re-examination of the esophagus and stomach failed to show mucosal transmigration despite special efforts (Fig. 5).

DISCUSSION

The first 4 cases illustrate prograde mucosal transmigration at the esophagogastric junction. A hiatal hernia of small size was demonstrated in each of these 4 cases. No other abnormalities were seen in the lower esophagus. Each of these 4 cases also exhibited transpyloric mucosal prolapse, an association which has been reported previously. The first 2 cases experienced symptoms related to the gastrointestinal tract, but in each of these a benign gastric ulcer was present, and there was no indication that the symptoms were related to the mucosal transmigration at the esophagogastric level. In Cases III and IV, mild gastrointestinal symptoms were experienced, but a positive relationship to mucosal transmigration was not established. Conceivably, in these 2 cases, the hiatal hernias could also have been responsible for the symptoms. In Cases I and II, the peptic ulceration was more likely responsible for the symptoms.

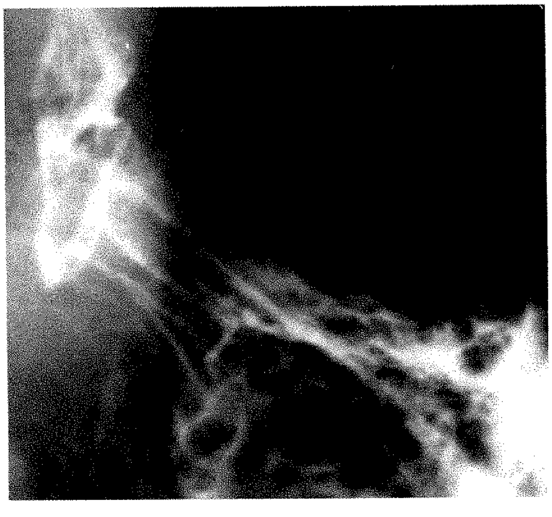

Fig. 3. Case III. Prograde transmigration.

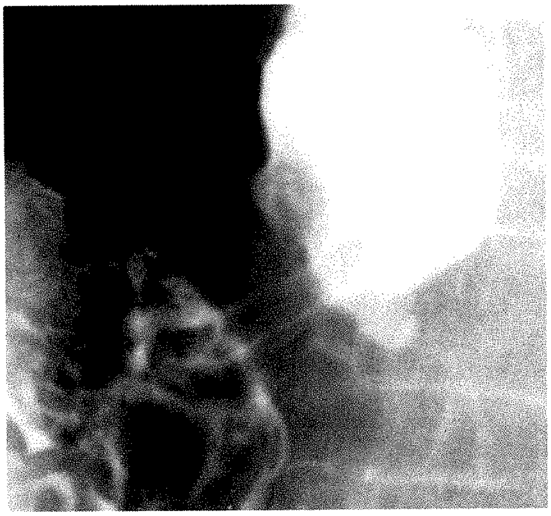

Fig. 4. Case IV. Prograde transmigration.

Case IV exhibited the typical "jack in the pulpit," or "arum lily" appearance previously described in other case reports. In the

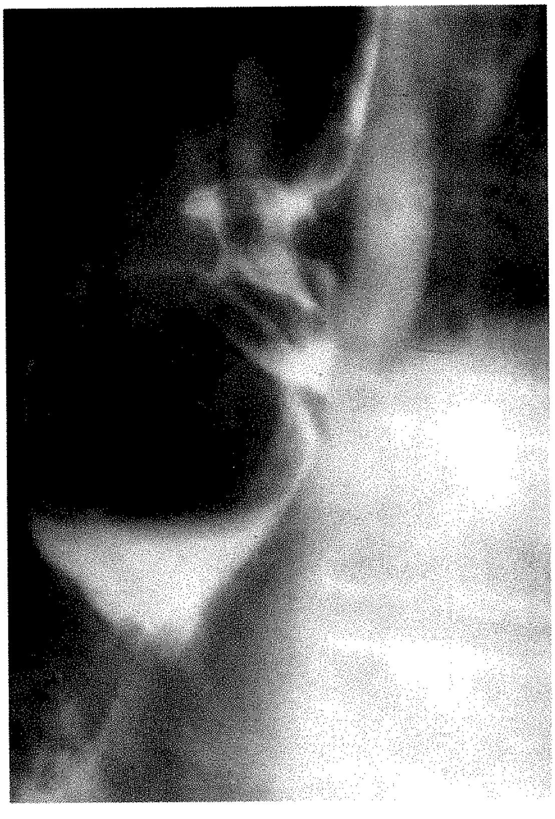

Fig. 5. Case V. Retrograde transmigration.

first 3 cases, less transmigration was present.

Only in Case v was there retrograde transmigration. In this patient, there was no transpyloric mucosal prolapse, but whether or not a relationship exists between its absence and the type of transmigration is questionable. This patient had the largest sliding-type hiatal hernia in this group. On re-examination the patient was asymptomatic and the transmigration could not be demonstrated. The transmigration might have been responsible for the symptoms, since the hiatal hernia was approximately the same size.

In demonstrating these changes, the recumbent positions, supine and prone, and Trendelenburg position were used in all cases. In Case v with retrograde prolapse, use of these maneuvers plus the return of the patient from recumbent to vertical position failed to demonstrate a deformity.

Although it has not been reported in Japan previously, these 5 cases of transmigration of esophageal mucosa were detected in approximately 2,500 consecutive gastrointestinal examinations. It is apparently associated with hiatal hernia and with transpyloric mucosal prolapse which is in accordance with reports in the literature of other countries. No case was detected in this series without a sliding-type hiatal hernia. All cases detected here have been in individuals 60 years of age and over. With special attention to the esophagogastric region, perhaps, more of these cases will be detected.

SUMMARY

A brief review of the English and Japanese medical literature pertaining to prolapse or transmigration of esophageal mucosa is given. Synopses of symptoms and clinical findings with illustrations of 5 cases in which this abnormality was detected are presented.

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RECENT ADVANCES IN THE REMOVAL OF MAGNETIC FOREIGN BODIES FROM THE ESOPHAGUS, STOMACH AND DUODENUM WITH CONTROLLABLE PERMANENT MAGNETS

By F. E. LUBORSKY,* B. J. DRUMMOND,* *and* ARTHUR Q. PENTA, M.D.†
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THE majority of foreign bodies that become lodged in the stomach and duodenum normally make an uneventful exit. There is, however, always the danger of intestinal perforation. This is particularly true of long, jagged and sharp pointed objects which have a prolonged sojourn in the stomach and when they fail to make the turn at the ligament of Treitz in the duodenal region. These should be removed as soon as possible.

In the past, before the development of high energy permanent magnets, the removal of foreign bodies from the stomach was usually a tedious procedure. Because the stomach is a hollow viscus and in constant peristaltic movement, foreign bodies usually shift in all directions. For their removal, it was necessary to employ esophagoscopy under biplane fluoroscopy before proper forceps application could be made. The entire procedure at its best was traumatic and time consuming. Then too, in many instances the patient became exposed to an overdose of radiation. Today, with the introduction of a magnet under fluoroscopic guidance offering no more difficulty than the passage of a Levin tube, the complexity of removing magnetic foreign bodies has been tremendously reduced.

The removal of foreign bodies by means of magnets is not a new procedure. Whalen,[7] in an excellent treatise on the subject, described the work of de Roaldes, who successfully removed a magnetic foreign body from the trachea by means of an electromagnet as early as 1900. Jackson,[3] in 1905, published a comprehensive analysis of the magnetic removal of foreign bodies from the food and air passages. In recent years, the development of the Alnico magnet[4,5] has given added impetus to this technique of foreign body removal. Equen,[1,2] one of the pioneers in the use of the Alnico magnet, has reported on a large series of foreign bodies removed with magnets.

Although the improved Alnico 5 magnet is quite strong, lifting almost one pound of weight in the sizes useful for these applications, failures in the magnetic extraction of foreign bodies are occasionally encountered. For example, when the contacting surface of the magnet with the foreign body is very small, the holding force becomes small. In these cases the foreign body may become detached at the cardiac and cricopharyngeus regions. The procedure in these rare instances requires the use of flexible grasping forceps to complete the extraction. In the removal of over 100 magnetic foreign bodies, forceps had to be employed only twice.

THE ON-OFF PERMANENT MAGNET

In the removal of magnetic foreign bodies, the permanent magnet has certain disadvantages. The prime disadvantage is that the operator has very little control over the portion of the foreign body contacted. For example, in the removal of an open safety pin from the stomach, it would be dangerous to attempt extraction unless the spring were contacted so that it was removed with the point trailing. Penta,[6] in 1946, tried to circumvent this difficulty by developing an electromagnet, whereby the magnetic field could be induced at the pleasure of the operator. This was a great improvement over the permanent magnet; however, it also had some disadvantages. Besides the obvious problems of maintaining a power supply and the danger of supplying electrical power to the coil inside the patient, the lifting force of the electromagnet was too small. Attempts to increase the magnetic force by increasing the power resulted in heating of the magnet to a degree unsafe for use.

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We will now describe a permanent magnet device which performs the same On-Off function as the electromagnet, yet has sufficient power to retrieve the foreign bodies encountered.

OPERATION AND CONSTRUCTION OF THE ON-OFF PERMANENT MAGNET

A photograph of this device and a schematic drawing of its active end are shown in Figure 1, *a* and *b*. The On-Off function is achieved by moving the Alnico 5 magnet out of its iron shield in the off position as shown, until it contacts the soft iron pole tip. It is then in the On position. The other end of the magnet remains shielded. In the Off position the flux from the magnet is shunted through the iron shield; in the actual design only 4 per cent of the total magnetic flux from the magnet is then present at the pole tip. The motion of the magnet is provided by a stainless steel cable soldered to the magnet and leading out to the finger grips. The spring provides necessary stiffness and smooth cable motion, while the plastic tube acts both to hold the magnet shield assembly and to provide the desired exterior surface.

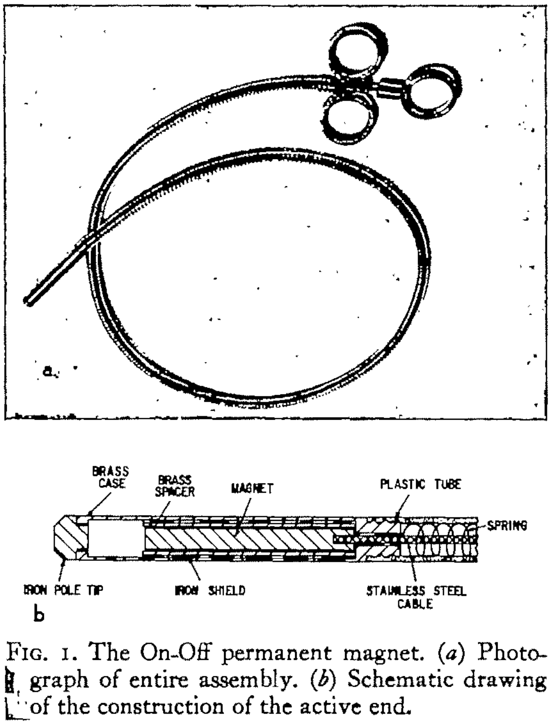

FIG. 1. The On-Off permanent magnet. (*a*) Photograph of entire assembly. (*b*) Schematic drawing of the construction of the active end.

The spacers, shield, pole tip and magnet length and diameter were designed for maximum lifting power, keeping in mind the space limitations of this application. Two sizes have been made. In the small, or children's size, the outside diameter of the magnet assembly is 0.25 of an inch and will lift a maximum of 160 gm. The larger, or adult size, has an outside diameter of 0.31 of an inch and will lift a maximum of 350 gm. These numbers may be compared to an electromagnetic unit with an outside diameter of 0.31 of an inch which would lift only a maximum of 100 gm. even when operated at its maximum power. This maximum power was determined by noting the power input needed to give a temperature rise of 60°F. in 1 minute in air.

It should be noted that the lifting forces quoted above are the maximum values achieved by lifting an object with a large flat face. Since the lifting force is proportional to B^2A, where B is the flux density through the contact area A, the lifting force will decrease as the surface contacted becomes smaller; for example, a curved or rough surface. The value of B is approximately constant since the pole tip is operating close to magnetic saturation. Thus, these same two magnets when contacted by the flat end of a $\frac{1}{16}$ inch diameter rod could

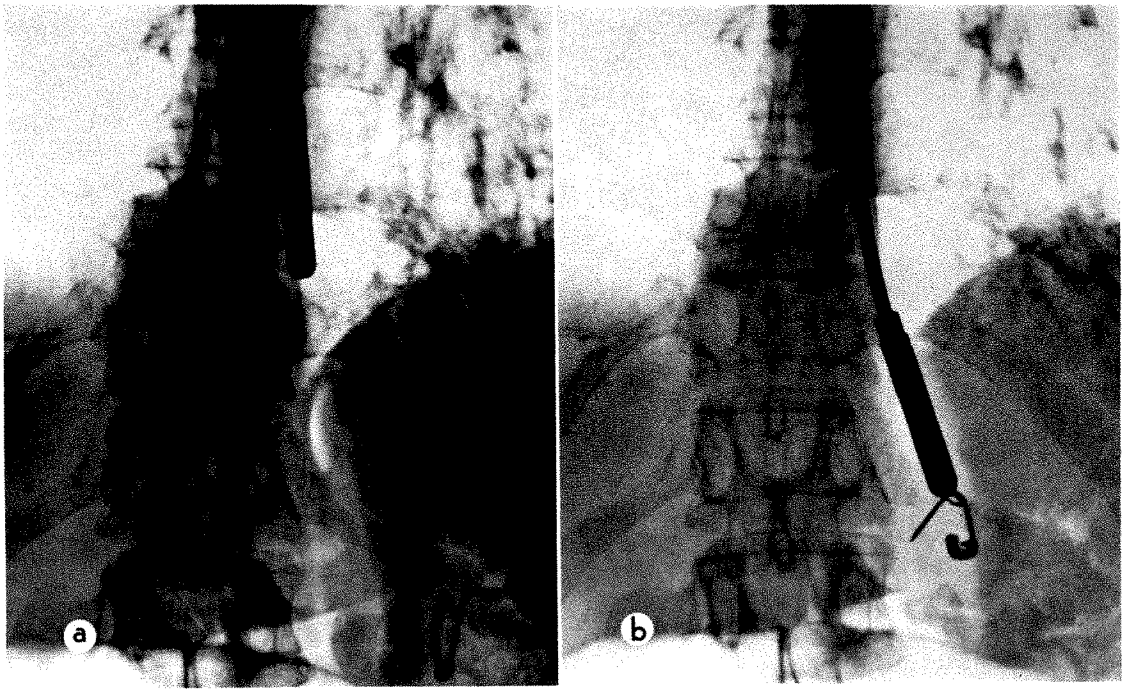

Fig. 2. (*a*) Roentgenogram showing the introduction of the On-Off magnet for the removal of an open safety pin, point upward, located in the stomach. (*b*) Roentgenogram showing the magnet attached to spring of pin and in the process of its removal. With a permanent type of magnet, the operator has very little control over the portion of the foreign body it is desirable to contact. However, with the On-Off magnet, the operator can control the magnetic field and thus make contact with the desired part of the pin.

raise only 130 and 210 gm. respectively, rather than 160 and 350 gm.

Roentgenograms are shown in Figure 2, *a* and *b* of the On-Off magnet before and after contact with an open safety pin. Note that it has been possible to contact the desired end of the pin.

STEERABLE PERMANENT MAGNET

Another problem confronting the endoscopist is the removal of magnetic foreign bodies from the stomach that become lodged high in the fundic region. Because of the peculiar angulation of the esophagus as it enters the stomach, any flexible tubing inserted into the esophagus, as it passes through the cardiac opening, will tend to fall away from this area of the stomach. To overcome this, it was necessary during the extraction to rotate the patient onto his stomach and to his sides. Even this technique was not always successful in maneuvering the simple magnet, attached to a flexible cable, into the fundic region.

A second permanent magnet device is next described which allows the physician to maneuver the magnet precisely to the point desired.

CONSTRUCTION AND OPERATION OF THE STEERABLE PERMANENT MAGNET

A photograph and schematic drawing of this device are shown in Figure 3, *a* and *b*. The bending action is obtained by pushing down on the control cable thumb grip. This pushes the magnet down until it is restrained by the control cord. Further motion then bends the stainless steel cable between the ends of the control cord, bringing the magnet tip up. The magnet may also be rotated by simply rotating the cable. The set screws at the control head can be tightened at any time to lock either or both the angular or rotational motion.

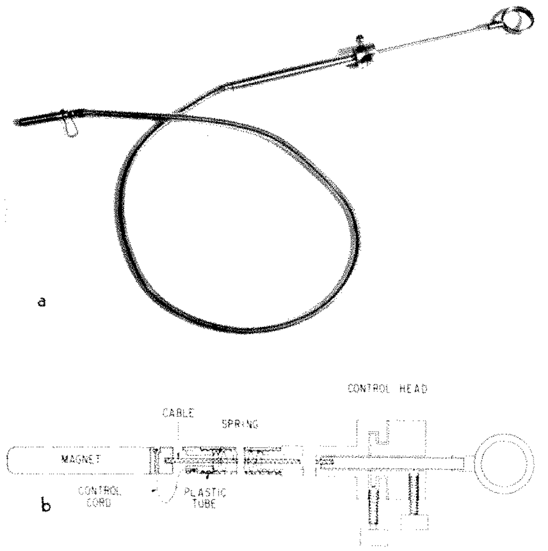

Fig. 3. The steerable permanent magnet. (*a*) Photograph of entire assembly. (*b*) Schematic drawing of its construction.

The cord, spring, and plastic tube provide the same functions as in the On-Off magnet.

This device has only been made with a 0.25 inch diameter Alnico 5 magnet. It will lift a maximum of 500 gm. or 240 gm. when tested against the $\frac{1}{16}$ inch diameter rod.

DISCUSSION

In many communities that do not have the service of a trained endoscopist, the general practitioner working under fluoroscopic guidance will soon become successful in the use of these magnetic devices. No anesthesia is required and the foreign body can be removed in 3 to 4 minutes without hospitalization of the patient. Introducing the magnet is a simple and safe procedure. It should be used early while the foreign body is still in the stomach. The quick and successful removal of the foreign body will eliminate the danger of perforation, hospitalization, and the repeated roentgenograms generally ordered to watch the daily progress of the foreign body as well as relieve the great anxiety on the part of the parents.

The question may be asked as to how one knows if the foreign body is magnetic. The majority of swallowed foreign bodies are in children. The parents usually bring along a companion to the object swallowed which can be tested beforehand. Where there is any doubt, a Berman locator can be used to detect whether the foreign body is magnetizable.

These new medical tools should be an important adjunct to those now available in the field of endoscopy.

CONCLUSION

Two new kinds of permanent magnet assemblies have been made and tested. One performs an On-Off function permitting an object to be picked up at any particular point, or released, if grasped incorrectly, and picked up again. The other device is a simple bar magnet which can be oriented in any direction allowing the physician to reach normally inaccessible regions. These two devices could also be combined into one unit.

The complete operation, from insertion of the device to its removal, may take only a few minutes. No anesthesia is required and the patient may be discharged immediately.

SUMMARY

The majority of magnetic foreign bodies that become lodged in the upper intestinal tract normally make an uneventful exit. However, there are instances where sharp, jagged or long pointed foreign objects, such as nails, tacks, bobby pins, needles and safety pins, because of their prolonged sojourn in the stomach or their failure to make the turn at the ligament of Treitz in the duodenum may cause intestinal perforation. The standard bar magnet, attached to a cable, is suitable for the removal of many, but not all, of these objects. Two new permanent magnet devices are described in this paper. The first device is a permanent magnet that can be turned on or off, necessary for cases in which the point of contact with the foreign body must be controlled. The second device is a steerable

permanent magnet which greatly facilitates reaching into normally inaccessible regions of the stomach. The introduction of these magnets under fluoroscopic guidance is a safe and simple procedure and offers no more difficulty than the passage of a Levin tube.

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GLOMUS TUMOR OF THE STOMACH*

A CASE REPORT

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GLOMUS tumor of the stomach is a rare entity. Perusal of the literature shows the first description by Kay *et al.*[6] in 1951. Since that time only 13 cases have been reported, none of which has appeared in the radiologic literature.[2,4,5,8–11,14–16] In reviewing the limited material, including the present case, certain salient points may alert the radiologist to consider this particular tumor in the differential diagnosis: (1) the majority of patients have been males; (2) there is a predilection for the antral portion of the stomach; (3) half the patients have presented with gastrointestinal bleeding; (4) roentgenographically, the tumor presents as an intramural extramucosal defect, with or without ulceration, and not infrequently of such soft consistency that changes in pressure alter its contour.

REPORT OF A CASE

This patient was a 67 year old white man admitted to the hospital in October, 1961, because of a movable left upper quadrant mass. He also complained of frequency, difficulty in voiding, and foul smelling urine. Other symptoms elicited were fatigue and constipation. The upper gastrointestinal series revealed a small tumor of the greater curvature margin of the gastric antrum (Fig. 1). It measured 3.0 cm. in diameter and varied in contour with palpation, at times being spherical and at other times ovoid (Fig. 2). Any margin could be altered by pressure, the tumor presenting a convex to flat surface. The overlying mucosa was effaced but not destroyed. The tumor formed an obtuse angle at its peripheral junction with the normal gastric wall. Normal peristalsis traversed the region. An excretory urogram and barium enema study were normal.

Laparotomy revealed a cyst of the inferior pole of the left kidney, accounting for the palpable left upper quadrant mass. The gastric tumor was submucosal and limited to the anterior wall of the distal antrum, 3.0 cm. proximal to the pylorus. Frozen section was reported as a malignant tumor, so a subtotal gastric resection was performed. Grossly, the tumor was solid, but soft, and had a tannish gray appearance with small focal areas of hemorrhage. The final microscopic sections revealed a typical benign glomus tumor rather than cancer. The tumor was composed of distended, irregular vascular spaces surrounded by proliferating sheets of cuboidal or polyhedral cells. These cells contained hyperchromatic nuclei with a clear perinuclear halo zone and eosinophilic cytoplasm. Penetrating finger-like projections extended between muscle bundles at the tumor margin, and there was no sharp demarcation or encapsulation. No mitoses were present (Fig. 3). The regional lymph nodes were normal.

Fig. 1. Spot roentgenogram of antrum showing the tumor with effaced but otherwise normal overlying mucosa.

The patient has been followed for 3 years without evidence of recurrence.

DISCUSSION

Normal glomus bodies are composed mainly of two elements, small arteriovenous anastomoses and a special type of epithelioid cells. The latter were first described by Masson[12] in 1924. Initially, it was felt that

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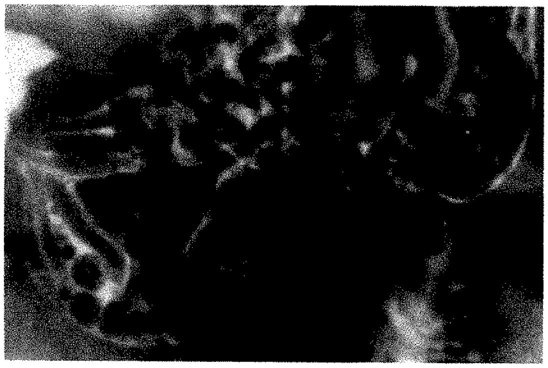

Fig. 2. Air contrast spot roentgenogram during the same examination as Figure 1 showing the alteration in shape of the tumor.

this epithelioid cell occurred only in the temperature-regulating glomus body and was limited to the hands and feet. Subsequently, Stout showed that the epithelioid cells were identical in morphology and behavior to the capillary contractile cell of Zimmerman.[17] Therefore, it serves as adventitial support and may contract the luminal width of the small vessels or capillaries.

Glomus tumors may occur anywhere in the body. However, they are not to be confused with tumors of the glomus jugulare (chemodectoma) or hemangiopericytoma, which are unrelated.[13] The location of most reported glomus tumors has been in the extremities, especially in the digits. While 14 cases have now been recorded in the stomach, interestingly, so far none has been recorded elsewhere in the gastrointestinal tract. With the epithelioid or contractile cell of Zimmerman present throughout the digestive tract, I am without explanation for this apparent selectivity.

Glomus tumors are usually encapsulated, but the capsule may be incomplete or absent as in the present case. Despite marginal infiltration, the biologic behavior of the tumor is benign. Since all the gastric

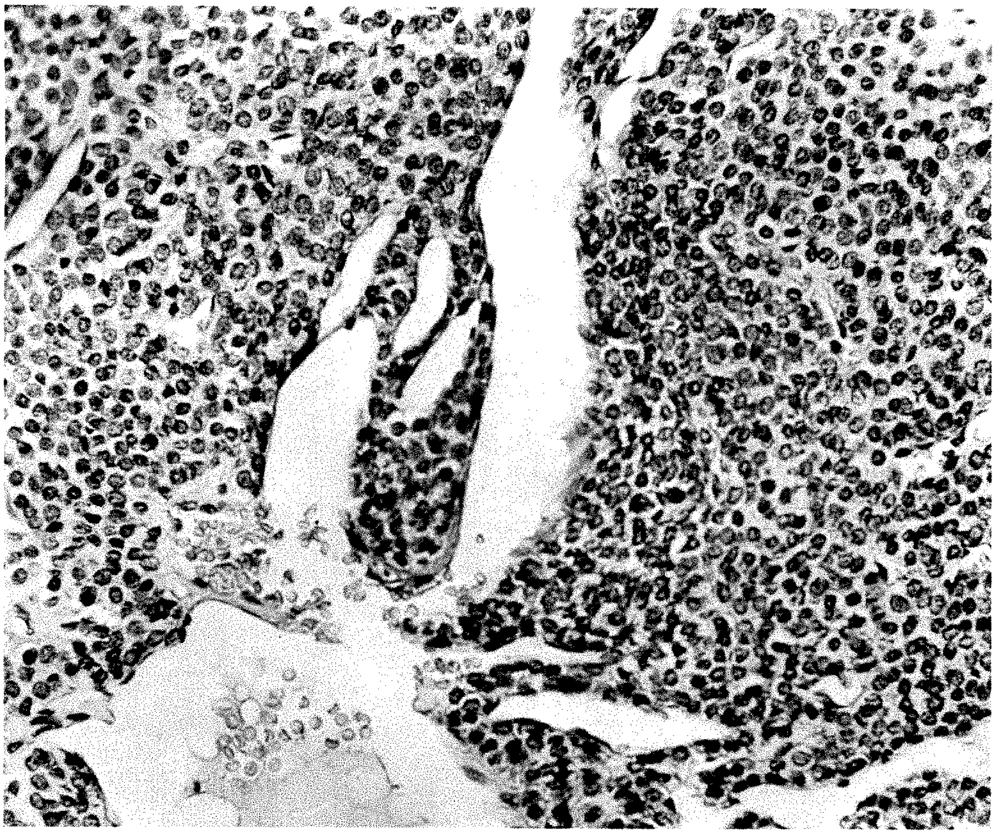

Fig. 3. Photomicrograph of the tumor showing capillaries surrounded by proliferating epithelioid cells (H & E stain).

glomus tumors have been submucosal, the roentgen characteristics have been those of other intramural extramucosal lesions, such as spindle cell tumors, ectopic pancreas, etc. The overlying mucosa may be effaced, but is otherwise unaltered. The margins are blunted with an obtuse rather than the acute angle one sees with mucosal tumors. Normal peristalsis is present and there is absence of fixation in the tumor area. A small mucosal ulceration has been present in the majority of gastric glomus tumors but, of course, may be found on occasion in any intramural extramucosal lesion. A most helpful roentgen sign is the changing shape of the mass, suggesting soft consistency. The presence of this particular finding narrows the possibilities to lipoma, hemangioma, or glomus tumor. Fat or calcified phleboliths, when present on the plain roentgenogram, would identify the first two lesions. The antral location and soft consistency of this lesion should bring this rare tumor to mind.

SUMMARY

Glomus tumor is another entity to be considered when one is confronted with a local intramural extramucosal lesion of the stomach. The roentgenographic demonstration of a soft tumor, with or without ulceration in a male, should suggest the possibility of a rare tumor as opposed to the more common intramural lesions. Glomus tumors are benign despite the absence, at times, of encapsulation.

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RETROPERITONEAL AND MEDIASTINAL CHEMODECTOMA*

REPORT OF A CASE AND REVIEW OF THE LITERATURE

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THE chemoreceptor system is composed of cells singularly adapted to respond to changes in arterial oxygen and carbon dioxide tension and pH by reflex control of the depth and rate of respiration.[44] These cells are formed into distinct structures, histologically similar to one another, scattered in the head, neck, and mediastinum. Mulligan[33] has coined the term chemodectoma (chemia—infusion, dechesthai—to receive, one—tumor) to describe tumors derived from the chemoreceptor system. These occur most commonly in the carotid body[21] and glomus jugulare.[42] Less frequently they have been noted in the vagus nerve and its ganglion nodosum,[22] in the paraganglion ciliare located in the orbit,[23] in the mandible,[6] the mediastinum,[22] the lungs,[12] intra-abdominally,[14] retroperitoneally,[8] and along the course of the femoral artery in Hunter's canal.[47]

The mediastinal chemoreceptors (aortic bodies, aorticopulmonary bodies, cardio-aortic bodies) were first described by Biedl and Wiesel[3] in 1902, and the first aortic body tumor was noted by Miller[43] in 1924. This early description appears to have been lost sight of in the more recent literature and Bloom[4] is usually given priority for the first notation of a mediastinal chemodectoma which he found, incidentally, in 2 male Boston terriers.

Normal chemoreceptor tissue has not as yet been demonstrated intra-abdominally in man but has been found in mice[16] and rats. Hollinshead[19] has noted that the abdominal bodies of mice may influence respiration when stimulated. Willis[52] suggests that chemoreceptor tissue is present in the retroperitoneal space of man but has not yet been demonstrated except as tumors.

It is the purpose of the author to report a case of multicentric chemodectoma occurring both retroperitoneally and in the mediastinum, in which the mediastinal lesion was delineated by thoracic aortography.

REPORT OF A CASE

The patient, a 24 year old white female, was first seen in the obstetrical clinic of the Kansas City General Hospital in June, 1955 when she was 3 months pregnant. Examination at that time revealed a uterus enlarged to the size of a 3 month gestation and a second, asymptomatic mass, located in the lower mid-abdomen and separate from the uterus, measuring 10 cm. in diameter. Retrograde pyelography performed shortly thereafter revealed a lower abdominal mass displacing the mid portion of the right ureter laterally but producing no obstructive hydronephrosis (Fig. 1).

Two months later, on August 3, 1955, she was admitted to the hospital. Physical examination revealed a normal progressive enlargement of the gravid uterus with no associated change in the second mass. Routine laboratory studies and chest roentgenograms were negative. A laparotomy was performed on August 11, 1955. The uterus was enlarged to the size of a 5½ month gestation. A retroperitoneal mass measuring 10 cm. in diameter was found, presenting slightly to the right of the midline overlying the bifurcation of the aorta and inferior vena cava. It was adherent to the common iliac arteries, the left iliac vein, and presacral fascia. The right ureter was displaced laterally but no abnormality of the kidney was noted. Numerous vascular channels, several 2 to 3 mm. in diameter, were noted crossing over the lobular glistening surface of the mass. There was no evidence of invasion of contiguous structures or of local lymph node metastasis. Attempts at sharp dissection of the mass met with profuse venous bleeding and only a biopsy was obtained. Pathologic examination of the

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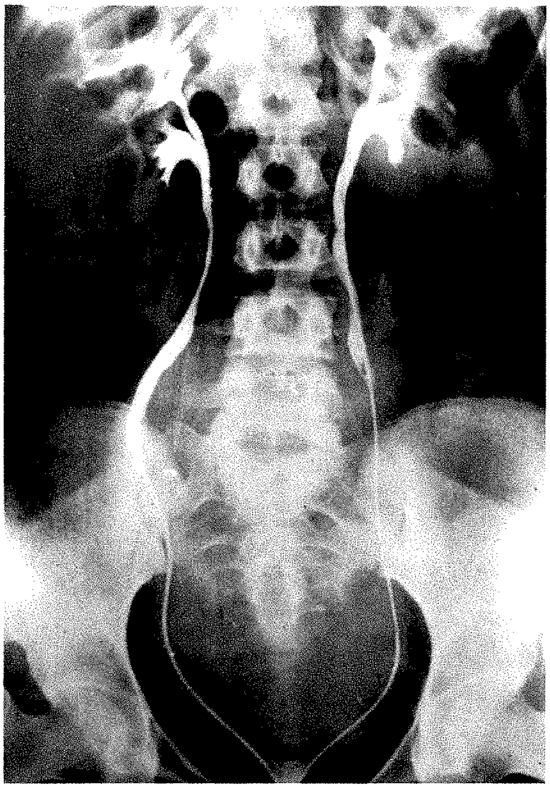

FIG. 1. Retrograde pyelogram showing the deviation of the right ureter by the retroperitoneal mass. A second mass in the pelvis is the gravid uterus. There is no evidence of obstructive hydronephrosis.

specimen revealed the presence of a chemodectoma.

On January 10, 1956 the patient was delivered of a normal 6 lb. 13 oz. female infant.

On February 9, 1956 a second laparotomy was performed. The mass had not changed in the interim and was removed *in toto* with moderate difficulty.

Grossly, the mass was lobular and encapsulated, measuring 9×9×4½ cm. It was pink, of rubbery consistency, and numerous vascular channels were crossing its external surface. Its cut surface revealed two opposing quadrantic areas of hemorrhagic necrosis. The remaining surface consisted of pink-brown tissue interlaced with thin fibrous bands (Fig. 2, *A* and *B*). Microscopy (Fig. 2, *C* and *D*) revealed an outer capsule of fibrous tissue with zones of hyalinization and endothelial lined vascular channels. The remainder of the sections consisted of large poorly defined cells containing large amounts of pale, eosinophilic, vacuolated cytoplasm and oval deeply stained nuclei. The cells were arranged in nests in many areas with thin fibrous strands and numerous dilated vascular channels interlaced between them. A diagnosis of chemodectoma (non-chromaffin paraganglioma), originating from the organ of Zuckerkandl, was made.

The patient was closely followed in the outpatient clinics because of many social problems. In October, 1956 she was seen because of severe left chest pain. Chest roentgenograms were reportedly negative and the pain disappeared in a few days after symptomatic treatment. During the intervening 5 years she complained intermittently of left chest pain. She was re-admitted to the hospital on April 10, 1961 because of severe incapacitating left subcostal pain of 1 week's duration. Physical examination revealed pain and tenderness over the left anterior 8th, 9th and 10th ribs. Chest roentgenograms (Fig 3*A*) were again reported negative, but the patient was treated clinically for fractured ribs. Following her discharge from the hospital she was seen at fairly regular intervals because of recurrent left chest pain. Repeated chest roentgenograms were interpreted as normal (Fig. 3*B*), although in retrospect some destruction of the left 8th rib posteriorly was noted.

On September 17, 1963 she was re-admitted because of paralysis of the lower extremities which had developed rapidly during the antecedent 12 hours. Physical examination revealed a well nourished, well developed patient complaining of inability to move her legs and severe left posterior chest pain. The vital signs were normal. Examination of the chest revealed an 8 cm. tender mass overlying the left 8th and 9th ribs posteriorly. Neurologic evaluation revealed no abnormalities of the cranial nerves or of the upper extremities. There was complete paralysis of the left lower extremity associated with anesthesia, analgesia, and loss of position and vibratory sense. The right lower extremity revealed slight residual motor function associated with hypalgesia and preservation of the 3rd–5th sacral segments. Knee and ankle jerks were absent bilaterally with positive Babinski reflexes. The sensory level extended to D8 on the left and D9 on the right. A lumbar puncture disclosed an opening pressure of 190 mm. of water. The Queckenstedt test was positive. The spinal fluid was clear, acellular, and con-

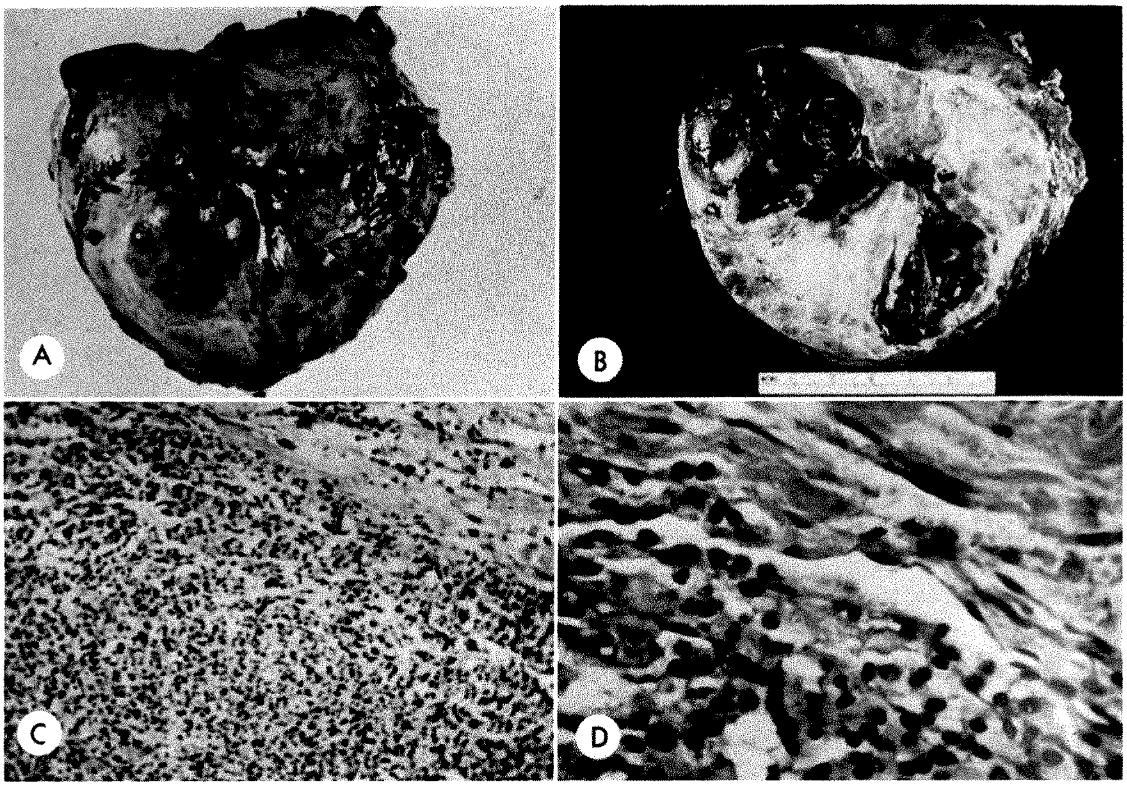

FIG. 2. Retroperitoneal chemodectoma. (*A*) Operative specimen showing its well defined capsule and lobulated surface. (*B*) Cut surface. Note the two large areas of hemorrhagic necrosis. The remainder of the mass is composed of pink-brown tissue. (*C*) Microscopic appearance. The cells are arranged in nests separated by interlacing bands of fibrous tissue and thin walled vascular channels (×100). (*D*) The cell borders are indistinct. The cytoplasm appears poorly stained and vacuolated whereas the nuclei are darkly stained and sharply demarcated (×430).

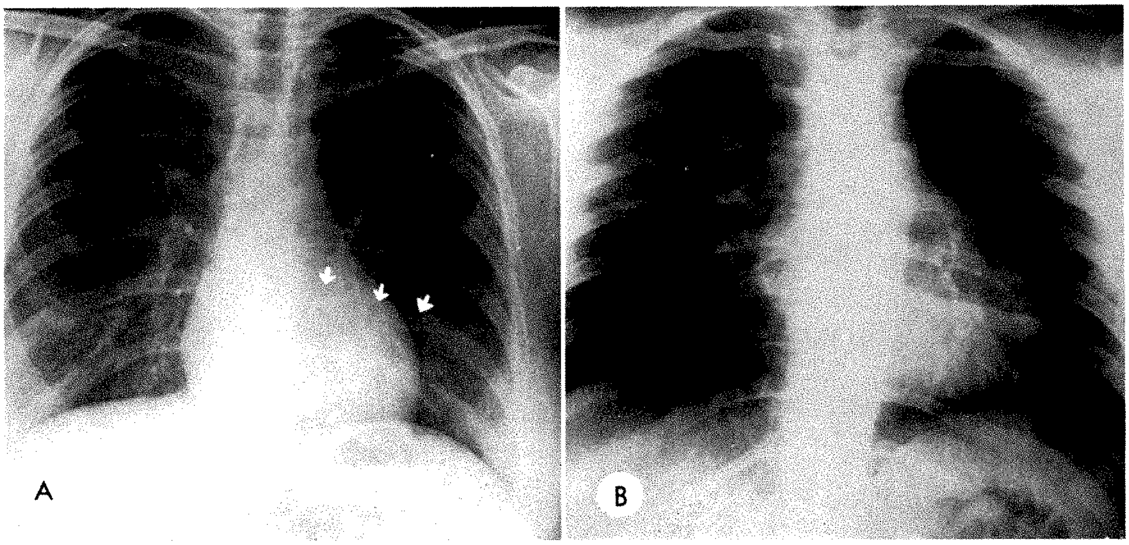

FIG. 3. (*A*) June, 1961. Destruction of the left posterior 8th rib can be faintly seen through the heart shadow. There is no widening of the paravertebral shadow. (*B*) December, 1962. Further destruction of the left rib. The left paravertebral shadow appears slightly widened at the level of D8.

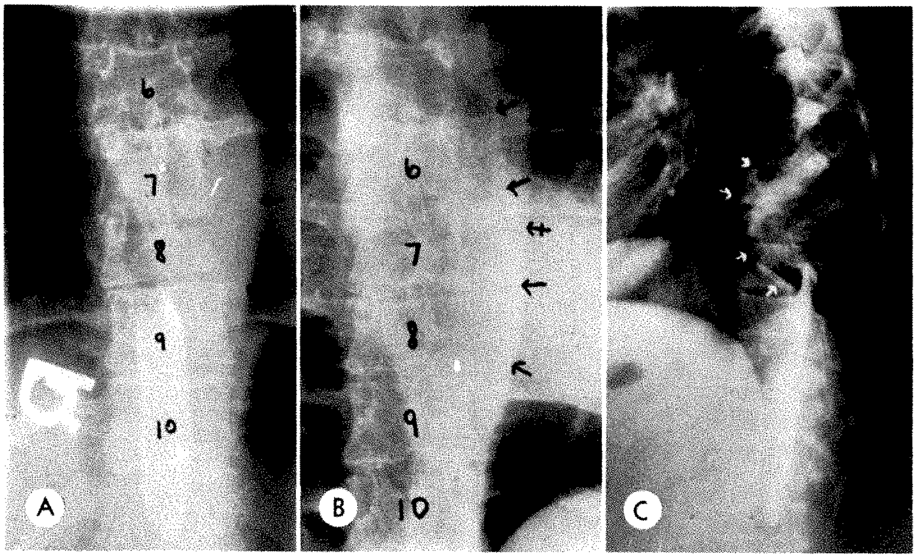

Fig. 4. (*A*) September, 1963. Myelogram demonstrating a complete block at the D8–D9 interspace. The 8th dorsal vertebra is collapsed and there is destruction of its pedicle. Note the bulge in the paravertebral shadow (arrows) produced by the tumor mass. (*B*) December, 1962. The 8th dorsal vertebra is not collapsed but there is destruction of its left pedicle. The bulge in the paravertebral shadow due to the posterior mediastinal mass (→) extends superiorly to the level of D6 where it is seen just within the shadow of the descending aorta (↛). (*C*) September, 1963. Lateral view demonstrating the block at D8–D9 interspace due to collapse of the 8th dorsal vertebra and extension of the tumor into the epidural space. The arrows outline the tumor mass.

tained 100 mg. per cent sugar and 200 mg. per cent protein.

Chest roentgenograms and spot studies of the dorsal spine revealed the presence of an 8 cm. rounded mass in the left posterior mediastinum overlying the 6th–9th dorsal vertebrae. There was associated destruction of the posterior portion of the left 8th rib and collapse of the 8th dorsal vertebra with destruction of its neural arch (Fig. 4, *B* and *C*). Immediate myelography revealed a complete block at the D8–D9 interspace (Fig. 4*A*).

An emergency decompressive laminectomy was performed within 3 hours after admission to the hospital. Tumor tissue was found surrounding the body of the 8th dorsal vertebra, destroying its spinous process and neural arch. The mass extended extradurally from D8 upward to D7. Because of profuse bleeding, only a decompressive laminectomy from D6 to D9 and a biopsy could be performed. The patient tolerated the procedure well but had little return of motor function.

The biopsy specimen revealed the presence of a chemodectoma similar in appearance to the one removed in 1956. Postoperatively, the patient had a course of roentgen therapy, receiving 3,500 r air dose in 3 weeks through a single 6 × 10 cm. port.

Following the termination of roentgen therapy, thoracic aortography was performed in order to evaluate changes in tumor size and vascularity. The Seldinger[15] catheter technique was utilized via a percutaneous puncture of the right femoral artery. The tip of the catheter was passed to the D4–D5 interspace. Forty cubic centimeters of 50 per cent hypaque was injected. The tumor mass was outlined but only a faint tumor stain noted (Fig. 5, *A* and *B*). Despite the apparent reduction in degree of vascularity of the tumor following irradiation, progressive destruction of D8 and involvement of D9 were noted.

The catheter was then withdrawn until its tip was at the L1–L2 interspace. A second injection of 30 cc. of 50 per cent hypaque was made. No evidence of residual or recurrent retroperitoneal tumor was noted (Fig. 6).

DISCUSSION

The chemoreceptor system, until recently, has been classified as part of the chromaffin or paraganglia system which

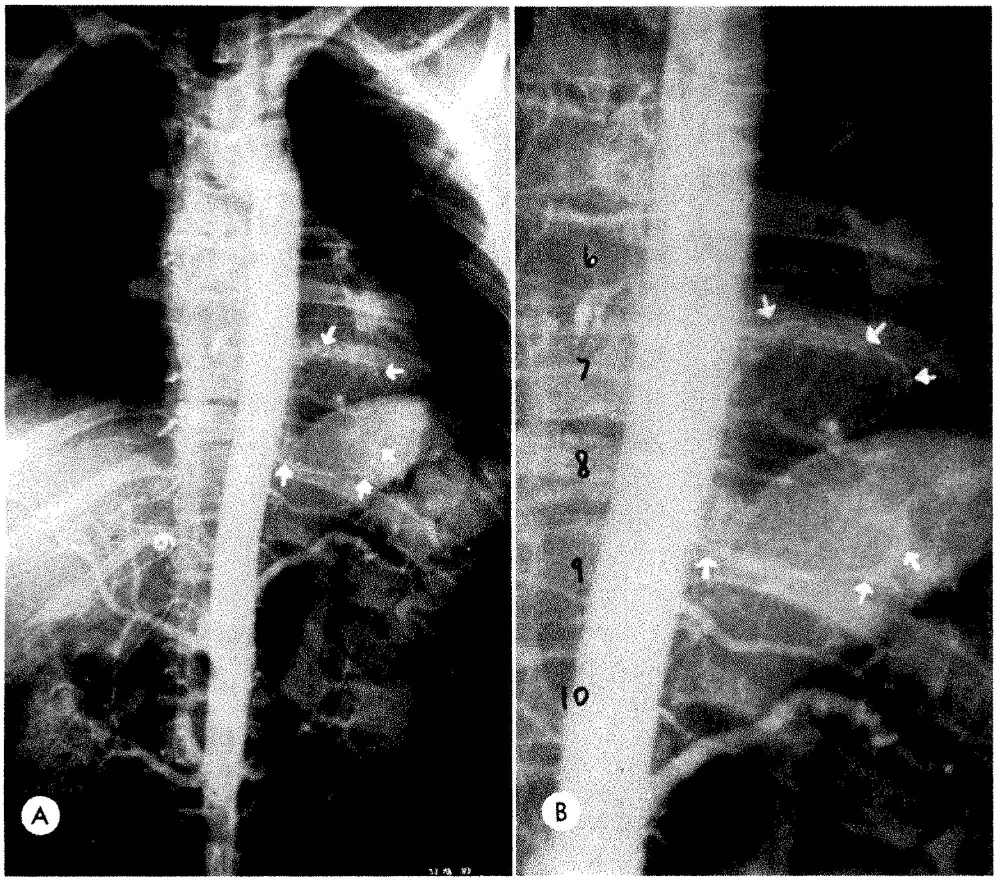

FIG. 5. (*A*) October, 1963. Thoracic aortogram made after a course of radiation therapy. A faint tumor stain due to filling of pathologic vessels by intercostal arteries arising at the level of D7 and D8 is seen. Another feeding vessel is seen outlining the lower edge of the mass at D9. (*B*) Magnified view showing a fine palisading of tumor vessels at the upper edge of the mass.

includes the adrenal medulla and nests of cells in the visceral sympathetic plexus ganglia along the thoracolumbar chain and in the celiac and pelvic plexus. Kohn,[20] who first postulated the paraganglionic theory, felt that these tissues originated from the neural crest and were characterized by a positive chromaffin reaction when fixed in fluids containing chromium ions.

As a result of the meticulous studies of Boyd,[5] de Winiwarter,[53] Nonidez,[37] Hollinshead,[19] and Comroe,[7] the structure and function of the chemoreceptor system has been more clearly defined, and the distinctive features which permit its separation from the chromaffin system have been elucidated. Structures classified as part of the chemoreceptor system should have the following characteristics: (1) show no evidence of endocrine function; (2) have a negative chromaffin reaction; (3) arise from neural anlage in close relation to the peripheral blood vessels; (4) have a sensory innervation; and (5) respond to changes of pH and CO_2 and O_2 tension in the peripheral blood.

Contrariwise, tissues of the chromaffin system should: (1) have an endocrine function characterized by the production of norepinephrine and epinephrine; (2) have a positive chromaffin reaction; (3) have a motor innervation; and (4) exhibit no chemoreceptor function.

The chemoreceptor system, concerned as it is with the maintenance of normal arterial oxygen tension, is phylogenetically

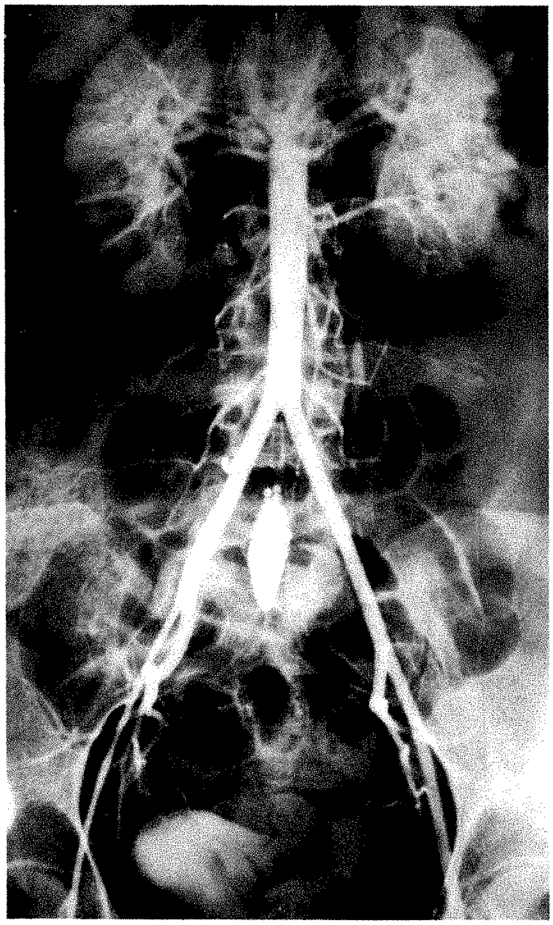

Fig. 6. October, 1963. Abdominal aortogram demonstrating no evidence of residual or recurrent retroperitoneal mass.

quite old. It is associated with the blood vessels supplying the gill clefts of primitive vertebrates, and it is therefore not surprising to find that the chemoreceptor system develops with the branchial arches in the human embryo.[5] The cells of origin are neuro-epithelial, originating from the various cranial nerves and migrating to sites of mesodermal condensation in the vessels of the primitive branchial arches.[1,5] The carotid body develops in relation to the third branchial arch and derives its sensory innervation from the glossopharyngeal nerve,[53] while the aortic bodies develop with the 4th and 6th branchial arches and are innervated by the sensory branch of the vagus nerve.[5,18]

The presence of chemoreceptor tissue in the retroperitoneal space can be explained by the coexistence of a cranial nerve (vagus) and the descending aorta. It is not surprising to find other derivatives of the branchial arches associated with chemodectomas. The alveolar body tumor of the mandible, reputedly a chemodectoma, is associated with the inferior alveolar artery, a derivative of the branchial arch, and the sensory branch of the trigeminal nerve.

The aortic bodies are composed of nests of "epithelioid" cells located in the middle mediastinum contiguous to the aortic arch and pulmonary arteries. These structures, 4 in number, have been described in the following locations:[7,36,37,38,51,53] (1) above the aortic arch near the origin of the right subclavian artery; (2) near the origin of the left subclavian artery on the lateral aspect of the aortic arch; (3) in the sulcus between the ascending aorta and pulmonary conus, near the origin of the left coronary artery; and (4) above and behind the bifurcation of the pulmonary artery, above the ductus arteriosus (Fig. 7).

Review of the literature reveals 16 previously reported cases of mediastinal[9–11,22,25,26,28,29,30,41,43,46,49,50] and 11 cases of retroperitoneal chemodectoma.[4,8,12,15,17,27,54] These cases and the presently reported one are summarized in Tables I and II.

Only a few of the aortic body tumors

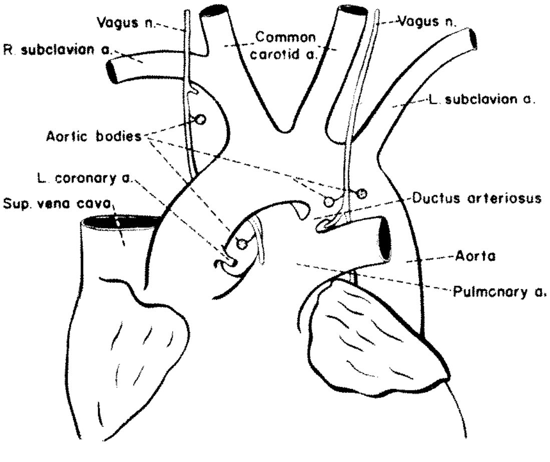

Fig. 7. The location of the normal aortic bodies. Modified after LeCompte.[24]

TABLE I

MEDIASTINAL CHEMODECTOMA

Case	Authors	Age and Sex	Signs	Symptoms	Location	Associated Chemodectomas	Roentgen Findings	Treatment	Comment
1	Miller 1932 (cited[43])				Right posterior costovertebral sulcus, level of 6th rib				
2	Lattes[22] 1950	59 M	Mass in right side of neck	Hoarseness, slight dysphagia, and dyspnea	Right anterior and middle mediastinum		Right mediastinal mass displacing the trachea and esophagus to the left	Surgery-biopsy	Alive and well
3	Lattes[22] 1950	35 F	Incidental finding at postmortem examination		At site of the obliterated ductus arteriosus	Carotid body ganglion nodosum			
4	Monro[30] 1950	22 M	Mass at left side of neck with enlarged cervical and supraclavicular lymph nodes	Pain radiating to back of left ear	Anterior mediastinum		Enlarged paratracheal lymph nodes; mottled calcified metastasis to the right lobe of the liver	Surgery-biopsy of cervical lymph nodes; roentgen therapy—4,000 r	Died with apparent metastasis to liver and cervical lymph nodes 8 years postoperatively
5	Duncan and McDonald[11] 1951	33 F	Routine chest roentgenogram		Right posterior costovertebral sulcus at level of D7		Mass in the right posterior costovertebral sulcus; 14 years later—destruction of D7 by the mass	Surgery-excision	14 years postoperatively developed transverse lesion of the cord with sensory level at D5 and spastic paraplegia
6	Duncan and McDonald[11] 1951	18 M	Routine chest roentgenogram		Right posterior costovertebral sulcus at level of 10th rib		Right posterior costovertebral mass producing erosion of the right 10th rib	Surgery-excision of a well encapsulated 7×5×4 cm. extrapleural mass	Alive and well
7	Drews and Groniowski[10] 1953	50 F	Decreased breath sounds in left chest	Left chest pain	Upper anterior mediastinum			Surgery-partial excision	Died of unknown causes 3 years postoperatively
8	McDonald *et al.*[28] 1954	38 F	Mass in right neck extending beneath clavicle	None	Right superior anterior mediastinum			Surgery-excision of a 7×7×3.5 cm. mass	Lost to follow-up
9	Davies and Randall[9] 1954	67 F	Incidental finding at postmortem examination		Overlying right auricular appendage				6×4×3 cm. mass
10	MacDonald[25] 1956	79 M	Incidental finding at postmortem examination		Encircling of left subclavian artery				5 cm. mass

(*Continued on the next page*)

TABLE I (*Continued*)

Case	Authors	Age and Sex	Signs	Symptoms	Location	Associated Chemodectomas	Roentgen Findings	Treatment	Comment
11	Shaw and Kennedy[46] 1956	30 M	Routine chest roentgenogram		Left posterior mediastinum at level of 9th intercostal space		10 cm. mass in the left posterior mediastinum	Surgery-excision of a 10×5 cm. mass	Alive and well
12	Mendelow and Slobodkin[29] 1957	31 F	None	Occasional sensation of heaviness in the retrosternal area	Right anterior mediastinum		Right anterior superior mediastinal mass displacing the trachea and esophagus posteriorly and to the left	Surgery-partial excision of a 12×5×5 cm. mass; roentgen therapy—2,400 r; the tumor was highly vascular, impinged on azygous vein and superior vena cava, and was fixed to the trachea	Alive and well
13	Taylor and Evans[50] 1958	13 M	Routine chest roentgenogram		Right posterior costovertebral sulcus at D6		7 cm. mass in the right posterior costovertebral sulcus		Died during induction of anesthesia; postmortem examination showed 7×5 cm. mass adherent to vertebral column
14	Madden[26] 1958	28 F	Routine chest roentgenogram		Right anterior mediastinum		7 cm. mass lying in contact with the right main stem bronchus	Surgery-excision of a 7×4×4 cm. mass	Alive and well
15	Tama *et al.*[49] 1962	66 M	Periorbital edema associated with engorgement of the veins of the neck and chest wall		Right anterior mediastinum		Venogram showed right anterior mediastinal mass producing block of superior vena cava	Surgery-biopsy and Teflon bypass; mass extended into lumen of superior vena cava; roentgen therapy—3,500 r	Alive and well
16	Phillips[41] 1963	58 M	None	Fatigue, shortness of breath, and intermittent numbness of the upper extremities	Left anterior and middle mediastinum		Left anterior and middle mediastinal mass displacing the trachea and esophagus to the right; aortogram delineated tumor and vascular stain	Surgery—6×7×4 cm. mass; only biopsy obtained due to severe blood loss	Died postoperatively
17	Haber 1964	24 F	Soft tissue mass overlying left posterior 8th and 9th ribs; paraplegia	Severe left chest pain; paralysis of lower extremities	Left posterior costovertebral sulcus at level of D8	Retroperitoneal in organ of Zuckerkandl	8 cm. left posterior mediastinal mass producing destruction of posterior left 8th rib and collapse of D8; aortogram delineated tumor and faint vascular stain	Surgery-decompressive laminectomy and biopsy due to severe blood loss; roentgen therapy—3,500 r	Alive 5 months postoperatively with persistent paraplegia

TABLE II

RETROPERITONEAL CHEMODECTOMA

Case	Authors	Age and Sex	Signs	Symptoms	Location	Associated Chemodectomas	Roentgen Findings	Treatment	Comment
1	Cragg[8] 1934	39 F	Incidental finding at postmortem examination		1. 3×2×1.5 cm. mass attached to anterior lateral surface of aorta just below origin of inferior mesenteric artery 2. 2.5×1.5×0.8 cm. mass just below the first mass	Carotid body			Died following surgery for a left carotid body tumor
2	Goodof and Lischer[15] 1943	47 M	Incidental finding at postmortem examination		1.5 cm. spherical nodule in the anterior portion of the body of the pancreas	Carotid body			
3	Smetana and Scott[47] 1951	23 M		Malaise, weakness and cough	Right lumbosacral area			Surgery-biopsy of an 20×12×6 cm. mass attached by a pedicle to an eroded area of the sacrum and the right psoas; roentgen therapy	Died 1 year postoperatively with metastasis to the lungs and adrenal
4	Smetana and Scott[47] 1951	27 F	Palpable, tender mass in the lower abdomen	Lower abdominal pain and dysmenorrhea	Attached to the lateral posterior abdominal wall			Surgery-excision of an 8 cm. mass invading the underlying abdominal wall	Local recurrence 1 year postoperatively; died 2 years postoperatively with clinical evidence of metastasis
5	Smetana and Scott[47] 1951	9 F	Lower abdominal mass		Attached to the left psoas muscle			Surgery-excision of an 8.3×6.5×5.5 cm. mass	
6	Smetana and Scott[47] 1951	39 F		Weight loss, indigestion, and intermittent vomiting	Above the right kidney			Surgery-excision of a 29×15×9 cm. mass	

(*Continued on next page*)

Table II (*Continued*)

Case	Authors	Age and Sex	Signs	Symptoms	Location	Associated Chemodectomas	Roentgen Findings	Treatment	Comment
7	Garvey and Claudon[12] 1953	21 M	Incidental finding at postmortem examination		3.5×5×6 cm. mass below the right renal pedicle and attached to the right lumbar sympathetic chain	Middle ear			Died following surgery for middle ear chemodectoma
8	Zacks[54] 1958	41 M	Incidental finding at postmortem examination		1. 7×6×4 cm. mass above and behind the 2nd duodenum 2. 1 cm. mass adjacent to larger one	Carotid body glomus jugulare			
9	Marshall and Horn[27] 1961		Incidental finding at postmortem examination						
10	Marshall and Horn[27] 1961		Incidental finding at postmortem examination						
11	Gough[17] 1963	42 M	Tender, pulsatile mass arising from the pelvis into the left hypogastrium	Pain in the left iliac fossa	Left pelvis		Arteriograms showed large ovoid tumor lying slightly to left of midline, displacing left iliac artery; tumor stain with network of irregular vessels	Surgery-excision of 9 cm. mass	
12	Haber 1964	16 F	Lower abdominal mass		Overlying bifurcation of aorta and inferior vena cava	Aortic body	Retrograde pyelogram showed displacement of right ureter; (3 month gestation); arteriogram performed 7 years later showed no evidence of tumor	Surgery-excision of 10 cm. mass	7 years postoperatively developed paraplegia from spinal involvement by a mediastinal chemodectoma

arise at the anatomic sites of the normal aortic bodies. These "aberrant" tumors may arise from nests of chemoreceptor cells along the intrathoracic part of the vagus nerve[50] or within the adventitia of the descending aorta.[11] Only 2 of the 17 cases of mediastinal chemodectoma had associated multicentric tumors, one of the glomus intravagale and carotid body, and the other of the organ of Zuckerkandl. In only 5 of the 13 operated cases could the mediastinal mass be completely excised, although in one of Duncan and McDonald's[11] cases there was an apparent recurrence 14 years later with destruction of D7 and symptoms of a transverse myelitis below the level of D5. A similar symptom complex developed in the present case 7 years following the onset of symptoms of left chest pain. Radiation therapy was attempted in 4 cases with no apparent regression of the tumor, although in the present case what was an extremely vascular tumor at the time of biopsy showed only a meager tumor stain on aortograms after a course of roentgen therapy. Only 1 case of distant metastasis was recorded.

Of the 12 cases of retroperitoneal chemodectomas, 5 had multicentric lesions and 2 had evidence of distant metastasis. It is of interest to note that the case reported by Goodof and Lischer[15] has been variously interpreted as an example of multicentric chemodectomas[54] and as a case of distant metastasis from a carotid body tumor.[6,35] In only 4 of the 6 operated cases could the mass be completely excised. The remaining 2 patients, one of whom received roentgen therapy, died within 2 years of surgery with evidence of distant metastasis.

Precise histologic criteria of malignancy have not been adequately defined for chemodectomas.[35] In view of this, malignancy must be assessed on clinical grounds—the presence of distant blood borne metastasis.[26] Even this criterion is faulty since chemodectomas are frequently multicentric in origin. Pettet *et al.*[40] have categorically denied the existence of proven metastasis from any chemodectoma. Le Compte[24] feels that most carotid body tumors are both histologically and clinically benign. Monro,[31] in a review of 179 cases of carotid body tumor, found 3 with evidence of distant blood borne metastasis. In a review of 90 cases of glomus jugulare tumor,[2] 3 cases demonstrated distant metastasis. Individual cases of metastasis to such sites as liver,[13] kidney,[48] heart,[32] pleura,[48] bone,[39] and skin[52] have been reported. To date, there does not appear to be any significant difference in the frequency of malignancy between the various homologous tumors of the chemoreceptor system.

Chemodectomas are slow growing tumors which are frequently asymptomatic until they have reached a large size. Although the ideal treatment would be complete surgical excision, this is frequently impossible due to the extreme vascularity of these tumors and their location contiguous to vital structures. Incomplete excision is compatible with a long survival. In Tama and co-workers[49] case, a Teflon bypass and radiation therapy successfully relieved superior vena caval obstruction caused by the tumor.

CONCLUSION

Radiation therapy has proven ineffective as the primary treatment of chemodectomas. The significant reduction in tumor vascularity following radiation therapy in the present case does suggest, however, that surgery following a course of radiation therapy may meet with greater success.

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THE INTESTINAL KNOT SYNDROME*

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ONE of the interesting variations in the spectrum of intestinal obstruction is the intestinal knot syndrome. Though a large variety of intestinal knot formations has been described, the syndrome usually consists of a twisting of the terminal ileum around the sigmoid colon which has in turn rotated on its own axis to create a volvulus (Fig. 1). The prognosis is very serious unless prompt surgical intervention is carried out.

The intestinal knot syndrome or compound volvulus has been described in northern Europe for more than half a century. Kallio[4] made a thorough study of the literature and reported 161 cases in 1932 which included the previously reported cases and 77 cases from Finland. Subsequently, intestinal knotting has been well described by Frimann-Dahl[1,2] who summarized the cases reported prior to 1942 and has since contributed other case reports and emphasized the roentgenologic features.

The intestinal knot syndrome has also been reported in other parts of the world. Recent case descriptions include a Persian male described by Schneider,[6] a Korean male reported by Mikal and Byers,[5] and cases in Bantu males described by Visser.[7] Also, White and Palmer[9] have discussed compound volvulus in a recent article on intestinal obstruction in Rhodesians. Although this entity is fairly common in Europe and Africa, it is apparently quite rare in this country, judging from the lack of case reports. Wangensteen[8] has mentioned the intestinal knot syndrome in his treatise on intestinal obstructions without detailed comment. A review of 52 cases of sigmoid volvulus treated at Grady Memorial Hospital over a period of 12 years revealed only 2 instances of intestinal knotting.

The anatomic basis necessary for an intestinal knot to occur is formed by a tortuous sigmoid colon with a long, freely mobile mesentery that is firmly fixed to the posterior abdominal wall and which serves as the axis for rotation. The ileum also has a long mesentery which allows free mobility and may lie either in front of or behind the sigmoid colon. Frimann-Dahl[1] postulates that there is a pre-existing mesenteritis with narrowing and approximation of proximal and distal sigmoid colon.

The etiology of this syndrome has not been determined. European authors have suggested that a bulky, high carbohydrate food intake may be responsible since this entity is seen in the lower economic group.

Intestinal knot formation most often appears in males in the fifth or sixth decades. There is abrupt onset of symptoms in a previously healthy individual with rapid progression to extreme prostration. The onset is characterized by violent persistent pain simulating an abdominal catastrophe such as mesenteric thrombosis or ruptured

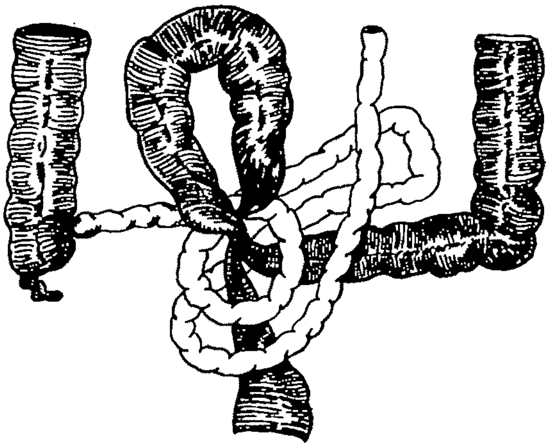

FIG. 1. Diagram demonstrating the mechanism of intestinal knot formation. For the sake of clarity, the resulting intestinal dilatation is not shown.

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viscus. Ileus develops rapidly and the abdomen becomes silent early, suggesting peritonitis. Usually, there is complete obstruction of both the ileum and sigmoid colon at the time of knotting, and following twisting of the mesentery, the blood supply is rapidly compromised with ensuing gangrene of the infarcted bowel.

The diagnosis of intestinal knotting can be made only with the aid of roentgenologic examination. In the supine roentgenogram of the abdomen, the dilated closed loops assume a characteristic pattern. The sigmoid loop projects upward and to the right. The obstructed colon tapers at the point of occlusion in a fashion characteristic of simple volvulus. The remainder of the large bowel contains a variable amount of gas and fluid, depending on the rapidity of the onset of obstruction and the duration of symptoms.

The obstructed and twisted ileal loops may be seen on the left side of the abdomen, so that the sigmoid and ileal loops are reversed in their usual positions. There is often considerable small bowel distention proximal to the ileal loop if obstruction is complete or nearly complete. The small bowel loops in the left lower abdomen may suggest mesenteric herniation, but this is ruled out by the dilated sigmoid loop. White and Palmer[9] stress that dilatation of the small bowel may be the most important single finding, since this is not seen in the early simple sigmoid volvulus.

If further confirmation is desired, a barium enema examination should be performed. The sigmoid shows a tapered end, typical of volvulus. In addition, Frimann-Dahl[1] has described a tapering off-shoot passing slightly beyond the "beak" around the encircling loops of small bowel, giving a "signet-ring" appearance in the sagittal plane.

At surgical exploration the dilated, gangrenous bowel loops are seen in the pattern demonstrated roentgenologically. Varying amounts of peritoneal fluid are found. Usually, resection of the involved large and small bowel will be necessary as the involved segments are gangrenous.

Due to rapidity of onset and severity of the disease, the intestinal knot syndrome carries a very poor prognosis. Early surgical intervention and adequate supportive therapy are the prime factors in patient survival.

The following 3 cases illustrate the pertinent clinical and roentgenologic features of the intestinal knot syndrome. All patients survived.

REPORT OF CASES

Case i. This 27 year old Negro male was admitted on July 26, 1957, approximately 12 hours after the onset of symptoms. On the morning of admission, he awakened with his abdomen tight and distended. There was progressive increase in the distention with nausea and vomiting. No flatus was passed on the day of admission, but the patient had a normal stool on the day before entering the hospital. He admitted considerable alcoholic intake on the previous night. There was no history of previous abdominal surgery.

On physical examination, the patient appeared acutely ill, and was exceedingly difficult to evaluate clinically. The temperature was 99.6°F., and the pulse rate was slightly increased. There was tenderness in both lower abdominal quadrants without rebound. No masses or hernias were noted. The bowel sounds were absent and the rectum was empty.

The roentgenographic studies (Fig. 2, *A* and *B*) showed a large loop of gas distended bowel representing the sigmoid colon in the right side of the abdomen. The proximal colon was not dilated. The dilated loops of ileum were present in the left lower abdomen. Air fluid levels were seen in the dilated intestinal segments in the upright examination. A chest roentgenogram showed no free intraperitoneal air or significant lung disease.

On abdominal exploration, there was a long redundant sigmoid colon which had wrapped itself around the base of a portion of the mesentery of the small bowel. Approximately 18 inches of the sigmoid and 9 feet of the small bowel were gangrenous. The peritoneal cavity contained about 200 cc. of dark bloody fluid. The surgical procedure consisted of resection

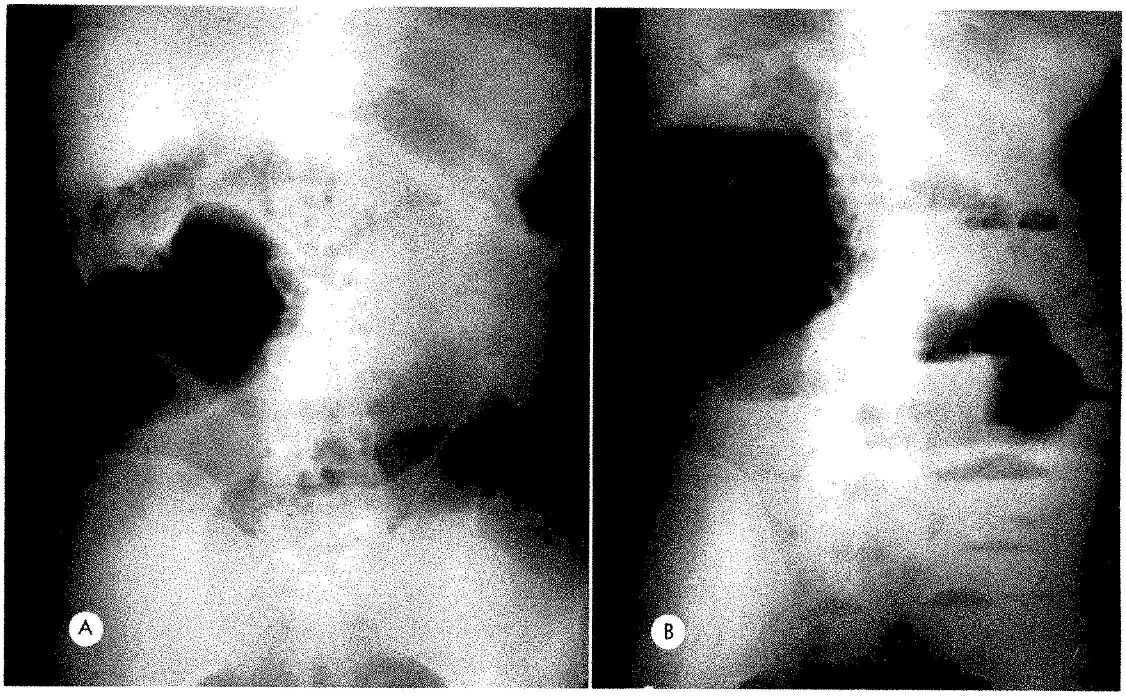

Fig. 2. Case i. (*A*) Supine and (*B*) upright roentgenograms of abdomen. A dilated loop of colon is seen in the right mid abdomen representing the twisted sigmoid loop. The small bowel is distended with multiple gas-filled loops in the left lower abdomen. Numerous air-fluid levels are present on upright roentgenogram.

of the gangrenous ileum and sigmoid colon and a primary entero-enterostomy for closure of the small bowel. A double-barrel colostomy was performed.

The postoperative course was uneventful and the patient was discharged. After an interval of 3 months, the colostomy was closed and the patient has remained well.

Case ii. This 51 year old Negro male was admitted on May 21, 1962 with symptoms of 10 hours' duration. He had experienced sudden onset of severe lower abdominal pain on the day of admission. There had been some alcohol and food intake on the day he entered the hospital. The cramping pain in the abdomen was severe and there was progressive abdominal distention. The patient had no bowel movements on the day of admission.

The physical examination revealed an acutely ill patient with severe abdominal pain. The temperature was 102°F. and the pulse rate was 120 per minute. The examination of the abdomen revealed generalized tenderness and muscle guarding. There was considerable abdominal distention and no audible peristalsis. The sigmoidoscopic examination did not reveal any obstructing lesion.

Survey roentgenograms of the abdomen showed a very large distended loop of sigmoid colon in the right lower abdomen (Fig. 3, *A* and *B*). The loops of the ileum in the left lower abdomen contained a moderate amount of gas. The walls of the ileum appeared thickened and there was little change in the position of these loops in the upright roentgenogram. On the erect examination, numerous air fluid levels were noted in the distended intestine. There was no free intra-abdominal air demonstrated.

On barium enema study only the segment of the colon distal to the sigmoid volvulus was demonstrated (Fig. 3*C*). There was an abrupt cut-off of the flow of barium to the distal area of obstruction where the contrast column assumed a tapered end.

The surgical exploration revealed a sigmoid volvulus beginning 8 inches above the peritoneal reflection. The twist was in a counterclockwise direction and all the bowel contained within this loop was gangrenous. The small intestine consisting of a portion of jejunum and ileum was knotted around the sigmoid volvulus

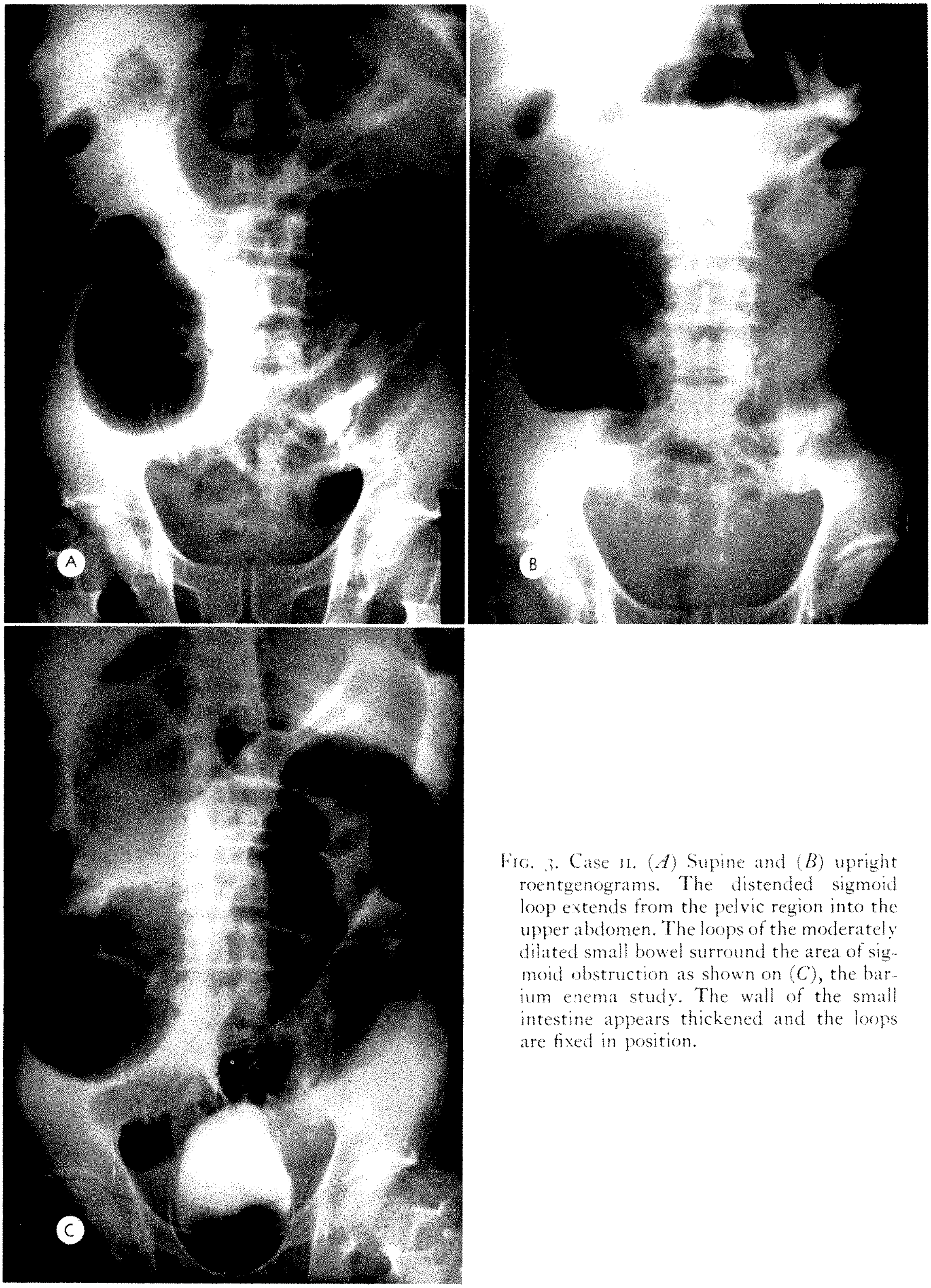

FIG. 3. Case II. (*A*) Supine and (*B*) upright roentgenograms. The distended sigmoid loop extends from the pelvic region into the upper abdomen. The loops of the moderately dilated small bowel surround the area of sigmoid obstruction as shown on (*C*), the barium enema study. The wall of the small intestine appears thickened and the loops are fixed in position.

and, in addition, had twisted on itself. The entire mid-portion of the small bowel was gangrenous. The superior mesenteric artery was patent with good pulsations into secondary arcades, and there was no evidence of mesenteric embolus.

The surgical procedure consisted of resection of the gangrenous sigmoid colon and small bowel. An entero-enterostomy and a double-barrel colostomy were done.

The colostomy was subsequently closed and the patient has remained well.

Case III. This 30 year old Negro female, eight months pregnant, was admitted to Coweta General Hospital on November 9, 1962. The patient had awakened on the day of admission with severe pain in the upper abdomen. A similar episode had occurred 4 weeks earlier, but had been less severe.

On physical examination the patient was found to be acutely ill and dehydrated. The temperature was 95.4°F., the pulse rate 88 per minute and the blood pressure 95/50. The abdomen showed marked tenderness in the epigastrium. The uterine size corresponded to the duration of pregnancy. Initially, fetal heart tones were heard in the left lower quadrant with a rate of 130 per minute. Pelvic examination revealed no effacement or dilatation of the cervix. The rectal examination was negative.

Approximately 12 hours after admission, the patient went into labor. The maternal pulse became quite rapid and the blood pressure could not be obtained. The uterus developed tetanic contractions and fetal heart tones could not be heard. The patient was exquisitely tender in the umbilical area. A stillborn male infant was delivered approximately 17 hours after admission. There was no evidence of retroplacental blood clot and the blood loss was minimal.

Roentgenograms of the abdomen approximately 10 hours after delivery showed a large, dilated loop of colon in the right upper abdomen and some small bowel loops in the left lower abdomen (Fig. 4, *A* and *B*). The lateral decubitus roentgenogram showed multiple air-fluid levels.

At operation, the sigmoid colon was found to be twisted on itself in volvulus formation. In addition, it had twisted around the small bowel causing the small bowel to twist on itself at the level of the superior mesenteric artery. The sigmoid colon was gangrenous and there was gangrene of the greatest part of the jejunum and ileum.

The sigmoid colon and 75 cm. of the small intestine had to be resected.

The postoperative course was stormy; however, the patient recovered and has done well.

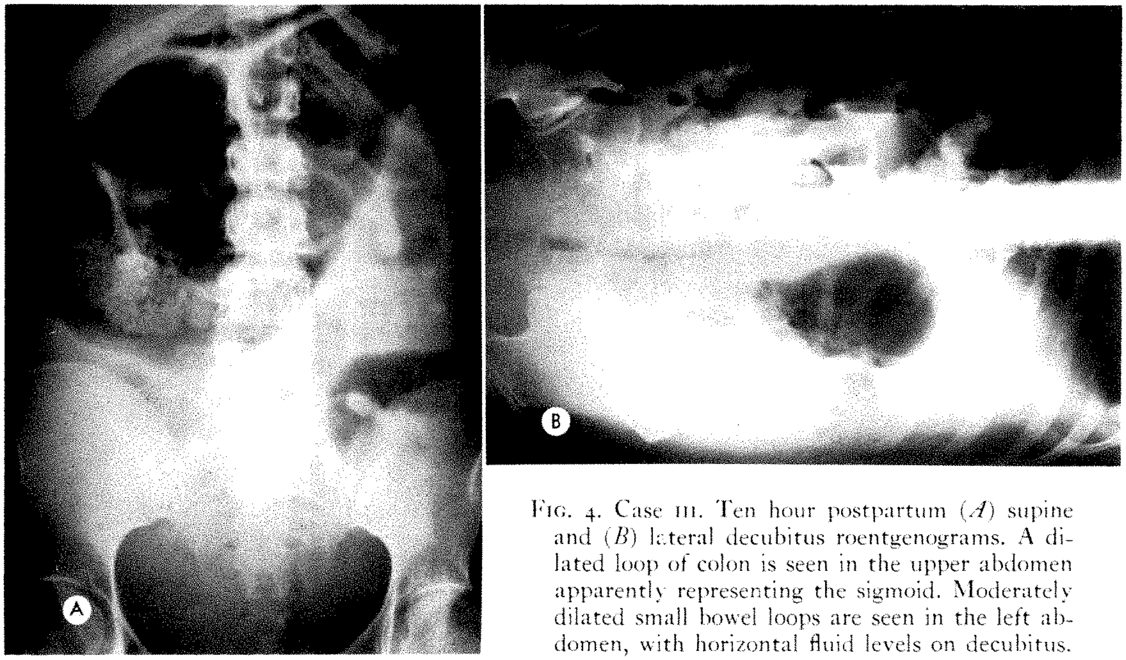

Fig. 4. Case III. Ten hour postpartum (*A*) supine and (*B*) lateral decubitus roentgenograms. A dilated loop of colon is seen in the upper abdomen apparently representing the sigmoid. Moderately dilated small bowel loops are seen in the left abdomen, with horizontal fluid levels on decubitus.

COMMENTS

The 3 cases described above showed several unusual features. Only 1 of the patients fell into the typical age range. The other 2 patients were younger than usually reported. One case was a female which is infrequently noted in any of the reported series. Otherwise, the patients showed clinical findings entirely consistent with those described in intestinal knotting. No definite etiologic factor could be established from the history of these patients. Two of the patients had alcoholic intake prior to admission, but it appears doubtful that this could be considered of etiologic significance in such a small series.

In the female patient, the intestinal knotting apparently precipitated fetal distress and premature labor, resulting in a stillbirth. Although the patient was quite ill at the time of admission, it was approximately 12 hours later that onset of labor occurred.

The roentgenologic findings in our cases were in accord with those described in the literature although the observations in the female patient were somewhat confusing due to the postpartum condition. In each case the dilated sigmoid colon occupied the right side of the abdomen and the distended ileal loops were found in a fixed position in the left side of the abdomen. A barium enema study was performed in only one case.

It is remarkable that all 3 patients survived as this condition carries a high mortality rate. Probably the younger age and prompt surgical intervention contributed to the favorable course in our patients.

SUMMARY

1. The intestinal knot syndrome consists of a twisting of the terminal ileum around the sigmoid colon which has in turn rotated on its own axis.

2. Though this condition has been commonly described in northern Europe, it has received little attention in the American literature.

3. Three cases are described which illustrate the pertinent clinical and roentgenologic features.

4. The following roentgenologic observations should suggest the presence of the intestinal knot syndrome:

a. Dilatation of the sigmoid colon with displacement into the right side of the abdomen.

b. The presence of dilated small gut loops, predominantly in the left lower abdomen, usually in a fixed position.

c. A "beaking" of the barium enema column at the area of the sigmoid volvulus with a tapering off-shoot around the encircling ileal loops giving a "signet-ring" appearance in the sagittal plane.

5. Without prompt surgical intervention, the prognosis is exceedingly grave.

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We wish to thank Drs. J. L. Clements, Jr., Joe Parks, and Jack Powell for contributing the third case.

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SMALL INTESTINAL DIVERTICULOSIS*

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NON-MECKELIAN diverticulosis of the small intestine, at one time considered a relative curiosity, has gradually achieved recognition as a significant clinical entity. A variety of secondary intra-abdominal complications has been recorded, and more recently an association with macrocytic anemia and steatorrhea has been emphasized. While the majority of patients with intestinal diverticulosis is relatively asymptomatic, there does exist the uncommon case with distressing symptoms of chronic low grade intestinal obstruction and progressive inanition and malnutrition.

HISTORICAL REVIEW AND INCIDENCE

The first description of small intestinal diverticulosis in the English medical literature is generally attributed to Sir Ashley Cooper, who reported in 1804 the postmortem findings in a 50 year old male dying of unrelated liver cirrhosis. From that time until 1923 only about 30 case reports appeared. In 1936, Rosedale and Lawrence[17] found an incidence of .06 per cent in a total of 5,000 necropsies, and the following year this incidence was increased to 1.3 per cent when they employed routine air insufflation of the bowel at the time of the postmortem examination.

The first roentgenographic demonstration in the living patient was by Case[3] in 1920; he reported 5 such cases out of a series of 6,847 small bowel examinations (0.073 per cent). In 1934, Rankin and Martin[13] diagnosed this lesion in 0.31 per cent of 956 consecutive small bowel studies. Two years later, in a series of 4,631 roentgen examinations, Edwards[5] recorded an incidence of 0.086 per cent, while, grossly at autopsy, he was able to demonstrate diverticula in 0.31 per cent (interestingly, a figure identical to that of Rankin and Martin). Ritvo and Votta,[16] in 1946, reported a 0.5 per cent incidence by roentgen examination and 0.42 per cent was given by Orr and Russel[12] in 1951. From these figures it would appear that one could expect to encounter small intestinal diverticulosis in less than 0.5 per cent of routine small bowel examinations.

AGE AND SEX

In contrast with the congenital variety of small intestinal diverticulum, which is generally found in the younger patient, the acquired type occurs in the latter decades. The majority of such patients will be between 55 and 70 years of age—the average being about 60.[1,2,15] Benson *et al.*[2] reported acquired intestinal diverticula in a patient, aged 90, and also one case in a child of 13 who was symptomatic for 9 years. Although some authors claim that there is no sex predilection, most series show a slight male preponderance in the ratio of 3:2 or 2:1.[1,2,9]

CLASSIFICATION

The earlier classifications of intestinal diverticulosis were based solely on the presence or absence microscopically of the normal muscular layer within the diverticulum. Thus, a so-called "true" diverticulum consisted of the normal four layers while the "false" variety, although containing mucosa, submucosa and serosa, showed an attenuated or virtually absent muscularis. The more recent classification divides them into the congenital and acquired forms: (A) Congenital: (1) Meckel's and (2) non-Meckelian; and (B) acquired: (1) primary (hernia) and (2) secondary (traction).

The majority of the congenital small bowel diverticula are of the Meckel's type, found in approximately 2 per cent of all adults. This diverticulum represents a failure of degeneration of the proximal yolk stalk, the remnant of which may extend to the umbilicus as a band or even more rarely as a duct. This pouch-like persistence is usually less than 10 cm. long and is found approximately 3 feet proximal to the ileocecal valve on the antimesenteric side of the intestine. The less common non-Meckelian type of congenital diverticulum most probably represents a variety of partial small bowel duplication, unrelated to the embryologic vitelline duct. Such a diverticulum may be cystic or tubular, single or multiple, and may or may not communicate with the intestinal lumen. Its exact origin still remains, for the most part, unsettled.[6,19]

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Both of these congenital varieties have the same anatomic structure as the remainder of the intestine, and thus will contain all four histologic layers. They usually occur in the younger patient on the antimesenteric border (although "true" duplications are found on the mesenteric border) and are usually solitary. They are larger than the acquired type and are found in the more distal portions of the gastrointestinal tract.

The acquired primary type of intestinal diverticulum is stated to be the most common lesion of the upper intestine. The colon is the most common site for intestinal diverticula followed by the ileum (Meckel's), duodenum, pharynx and esophagus, stomach and jejunum. In the small intestine, exclusive of the duodenum, diverticula are most often found in the age group of 45–70 years, mainly in the proximal portion of small bowel near the ligament of Treitz. There is a diminution in size and number distally in the small bowel. Diverticula are uncommon in the ileum, and when found here are usually in the terminal segment, are small and inclined to be multiple. A single, isolated acquired diverticulum is most uncommon; multiple diverticula are the general rule. As many as 400 diverticula have been reported in a single case.[7]

Associated diverticula are frequently found in other parts of the gastrointestinal tract, most commonly in the colon and duodenum. Depending on the series reported, the figures range from 24 per cent to 53 per cent for associated colonic diverticula and 22 to 44 per cent for duodenal diverticula.[1,2,10,15]

The acquired primary type of intestinal diverticulum is found on the mesenteric border of the intestine at the site of vascular penetration, and may vary from a few millimeters to 10 centimeters in size. Histologically, there is a thinned or absent muscular coat; these diverticula represent a type of mucous membrane herniation through this muscular defect.

The less common secondary form of acquired intestinal diverticulum originates from contiguous inflammatory disease with consequent traction on the bowel wall, *i.e.*, traction diverticulum.

PATHOGENESIS AND ETIOLOGY

As noted previously, the primary type of acquired diverticulum is produced as a result of mucosal herniation through a weakened or attenuated layer of muscularis. While no single factor is responsible, it has been shown that a weakened area exists in the intestinal musculature at the site of penetration by the mesenteric vessels. In his classic monograph, Edwards[5] showed that the paired mesenteric vessels pierced the muscularis to reach the submucosa in such a fashion as to produce fairly large defects in the circular and longitudinal layers, which are almost directly in line with one another. This localized area of structural weakness, the so-called "locus minoris resistentiae," together with the usual sparsity and weakness of the longitudinal muscle layer on the mesenteric side of the upper intestine, is generally accepted as the major inherent predisposing factor. The additional factor of increased intra-abdominal pressure, whether sudden as with straining or coughing, or of a more chronic nature secondary to "peristaltic irregularity," chronic constipation, or overdistention, seems to play a significant contributory role.

Degenerative disease due to a variety of causes such as senile atrophy, vascular sclerosis, fatty infiltration, venous stasis and local irritation has also been mentioned as an etiologic factor. In addition, the theories of congenital hypoplasia, toxic myositis, and obesity with an increase in mesenteric fat acting as a "leader" for herniation have variously been considered, only to be subsequently discarded.

CLINICAL MANIFESTATIONS

The clinical spectrum of small intestinal diverticulosis is quite broad and diverse, ranging from the asymptomatic patient through chronic deficiency states to an acute intra-abdominal catastrophe. There are 4 major clinical manifestations of this entity: (1) no apparent symptoms; (2) chronic dyspepsia and abdominal discomfort with or without low grade obstruction; (3) complications requiring surgery; and (4) steatorrhea and/or macrocytic anemia.

NO APPARENT SYMPTOMS

The majority of any series of patients with small intestinal diverticulosis is probably asymptomatic or at the most complains of symptoms that are difficult to relate to this condition.

Some authors feel that the higher incidence of symptoms in diverticulosis of the duodenum as compared with the jejunum is related to the fact that the jejunum is not fixed as is the duodenum, but rather free and mobile; thus the jejunal sacs are able to empty. However, the over-all incidence of duodenal diverticula is 5 times as frequent as diverticula in the jejunum, possibly accounting for this greater frequency of associated symptoms.[10]

CHRONIC DYSPEPSIA WITH OR WITHOUT OBSTRUCTION

A variety of chronic gastrointestinal symptoms has been ascribed to small intestinal diverticulosis. Colicky pain, dyspepsia, abdominal discomfort, loud rumbling noises emanating from the abdomen, flatulence, bloating, diarrhea, constipation, postprandial nausea and vomiting, anorexia and weight loss all have been described. These symptoms seem to have no relation to bowel movements, exertion or meals, and are usually intermittent. In many such patients a careful clinical evaluation often discloses concomitant conditions that more satisfactorily explain the symptom complex; thus other entities must always be excluded when small bowel diverticula are found.

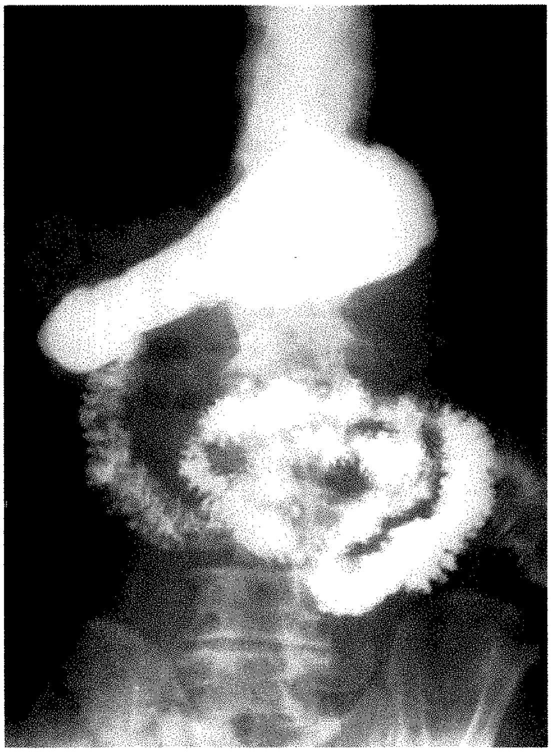
There does remain a group of patients who presents with a variety of such symptoms and in whom no other cause is apparent. There are recurrent bouts of chronic low grade intestinal obstruction varying from mild discomfort to disabling symptoms, necessitating surgical intervention. At surgical exploration in such patients, the bowel wall may be found to be thickened, dilated and incapable of efficient peristalsis. While the underlying mechanism is not completely understood, it is felt that the jejunal wall, weakened by multiple mucosal herniations, fails to participate in effective peristalsis, with resultant hypertrophy and progressive decrease in contractility.

SURGICAL COMPLICATIONS

Severe life threatening complications requiring surgical intervention are not common but occur with sufficient frequency to merit serious consideration, especially in a patient with known diverticulosis of the small intestine. These include: (a) intestinal obstruction (acute); (b) inflammation; (c) hemorrhage; and (d) rupture. These complications may occur singly or in combination but are usually interrelated.

(a) Acute intestinal obstruction is the commonest complication[10] and may be produced in a variety of ways such as intussusception, volvulus or postinflammatory adhesions. A retained enterolith or foreign body may be associated[20] and acute diverticulitis may occur and result in localized abscess, stricture and adhesions or perforation and generalized peritonitis.[9,14]

FIG. 1. Case 1. Anteroposterior supine roentgenogram of the abdomen with barium in the stomach and proximal jejunum shows scattered, small diverticula arising from the proximal jejunum just distal to the ligament of Treitz.



(b) Acute inflammation is said to be uncommon because of the relatively wide necks of the diverticula and the fluidity and sterility of their contents.[1] A concretion within a diverticulum causing acute inflammation is quite rare but has also been reported.[16]

(c) Hemorrhage, when present, is usually manifested in the form of melena, although reverse peristalsis may result in hematemesis. Such hemorrhage can be massive in amount, and Kozoll *et al.*[8] report on a patient who prior to operation lost 16,600 cc. of tarry stool, and on surgical exploration was found to have several diverticula con-

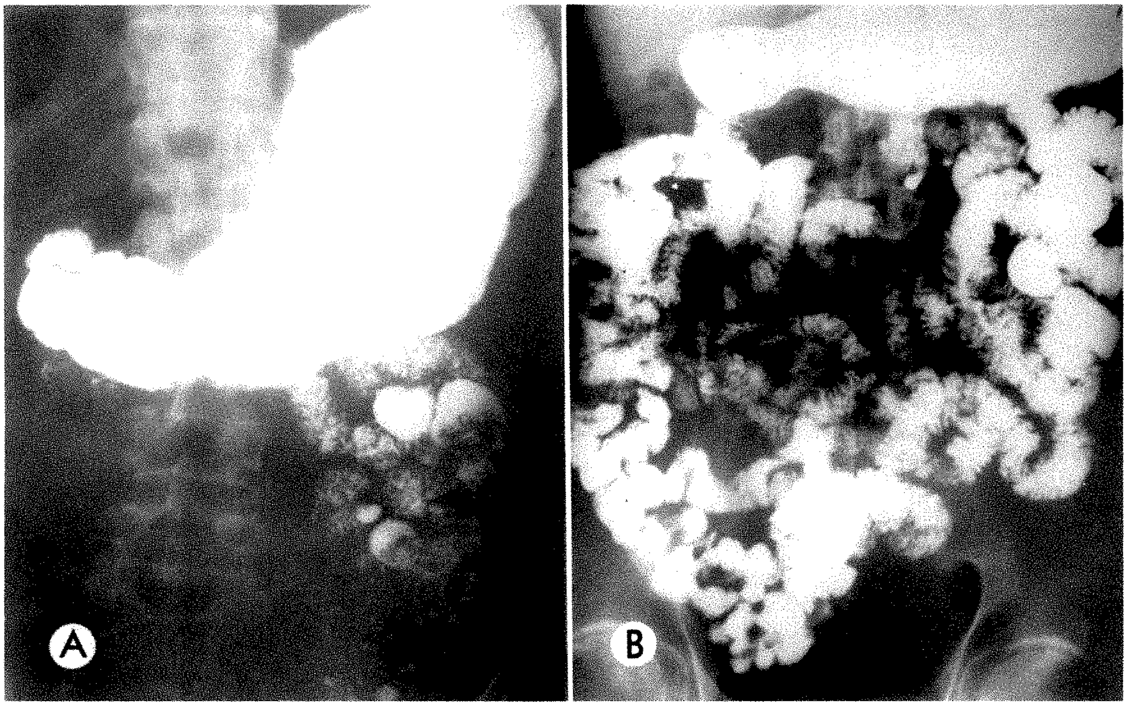


FIG. 2. Case 3. (*A*) Posteroanterior prone roentgenogram of the abdomen with barium in the stomach and proximal jejunum demonstrates several fairly large, wide-mouthed diverticula arising from the proximal jejunum distal to the ligament of Trietz. (*B*) Posteroanterior prone roentgenogram of the abdomen with barium outlining loops of the jejunum and ileum, and ascending colon again shows the large diverticula confined primarily to the proximal jejunum.

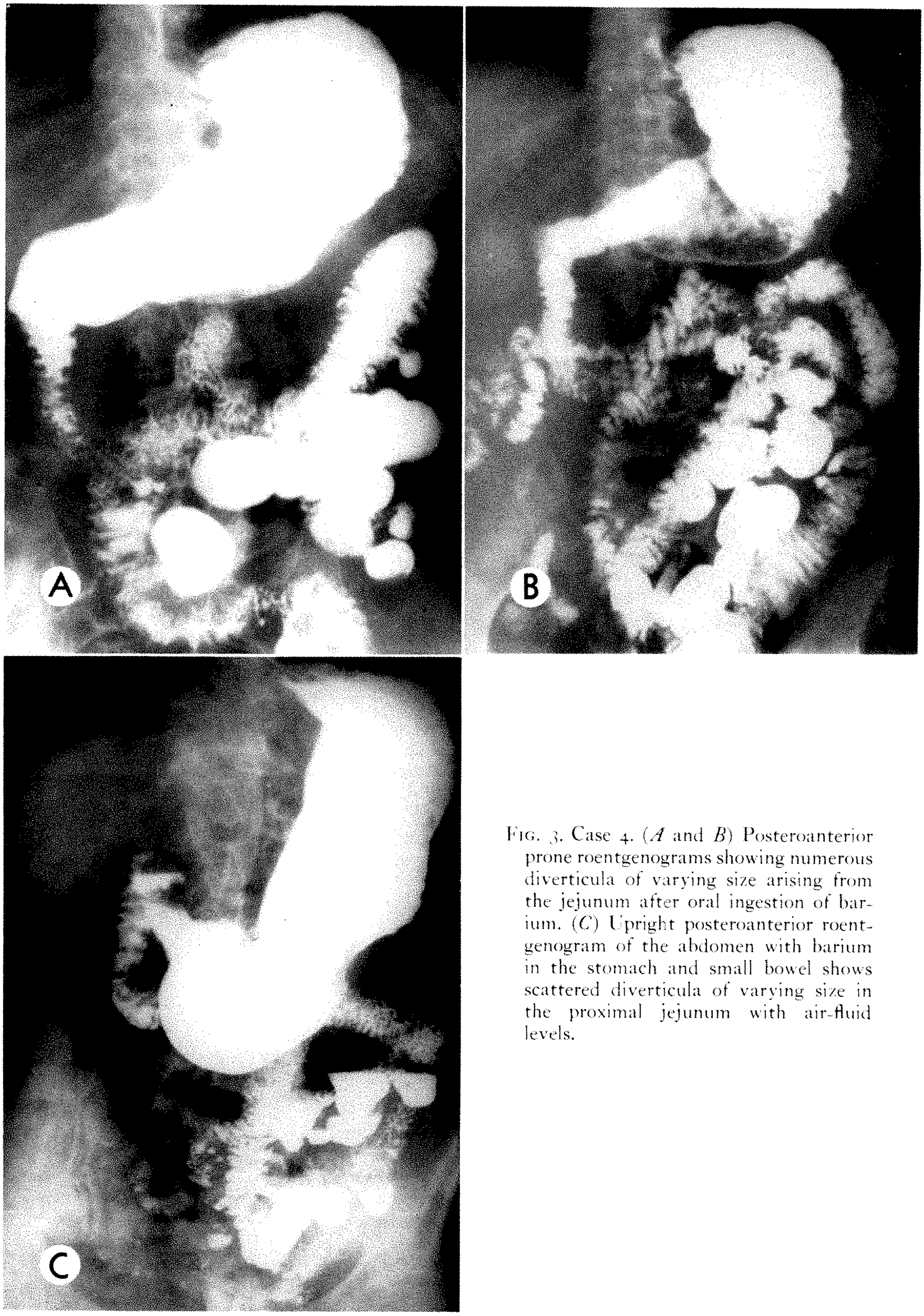


FIG. 3. Case 4. (*A* and *B*) Posteroanterior prone roentgenograms showing numerous diverticula of varying size arising from the jejunum after oral ingestion of barium. (*C*) Upright posteroanterior roentgenogram of the abdomen with barium in the stomach and small bowel shows scattered diverticula of varying size in the proximal jejunum with air-fluid levels.

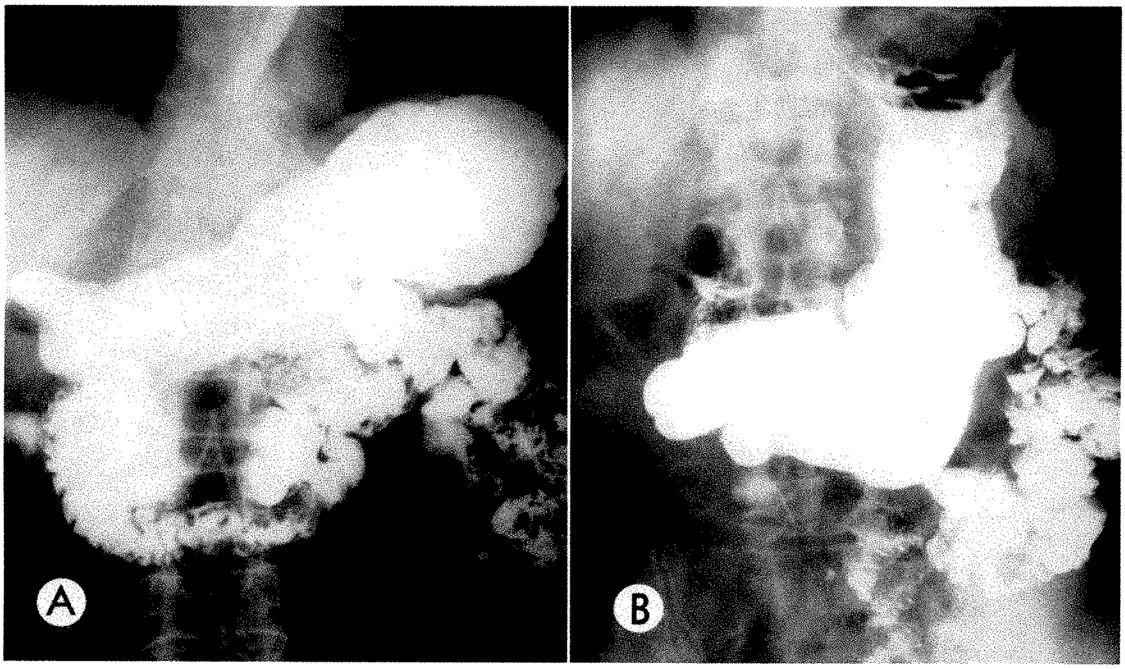


Fig. 4. Case 6. (*A*) Anteroposterior supine roentgenogram of the abdomen with barium outlining the stomach and proximal jejunum demonstrates numerous diverticula of varying size arising from the proximal jejunum. Several of the diverticula have a relatively narrow mouth. (*B*) Erect posteroanterior roentgenogram with barium in the stomach and proximal jejunum delineates several air-fluid levels within diverticula of the proximal jejunum.

taining small ulcerations. An innocuous appearing diverticulum may be easily overlooked at the time of exploratory laparotomy and in reality be the site of such bleeding, necessitating reoperation. The cause of such bleeding is not always determined, although such patients usually remain well after segmental resection.[1] Mucosal ulceration, heterotopic rests, tearing of vessels by distention of the sac, external trauma and trauma secondary to concretion and foreign bodies have been mentioned as causative factors of hemorrhage.[7,8,12,19,20]

(d) Rupture of a diverticulum may be related to the presence of inflammation enteroliths, foreign bodies, trauma, vascular compromise or even parasites.[2] Spontaneous pneumoperitoneum without peritonitis has been reported.[11]

Various neoplastic lesions have been described within diverticula, and are usually asymptomatic until late. Benign tumors include heterotopic pancreatic rest, fibroma and lipoma.[2] A case of primary jejunal carcinoma within a diverticulum has been reported.[2]

STEATORRHEA AND/OR MACROCYTIC ANEMIA

During the last decade the association of macrocytic anemia and steatorrhea with small intestinal diverticulosis has become recognized. Although the majority of such cases has presented with this triad, there are patients who solely manifest steatorrhea or impaired absorption of vitamin B_{12} with or without associated macrocytic anemia. The experimental construction of diverticula in rats with production of macrocytic anemia suggests the importance of stasis and stagnation within the blind pouch, and a consequent alteration in the normal intestinal bacterial flora.[18] The exact mechanism of such interference with absorption is not known with certainty. However, the marked clinical improvement

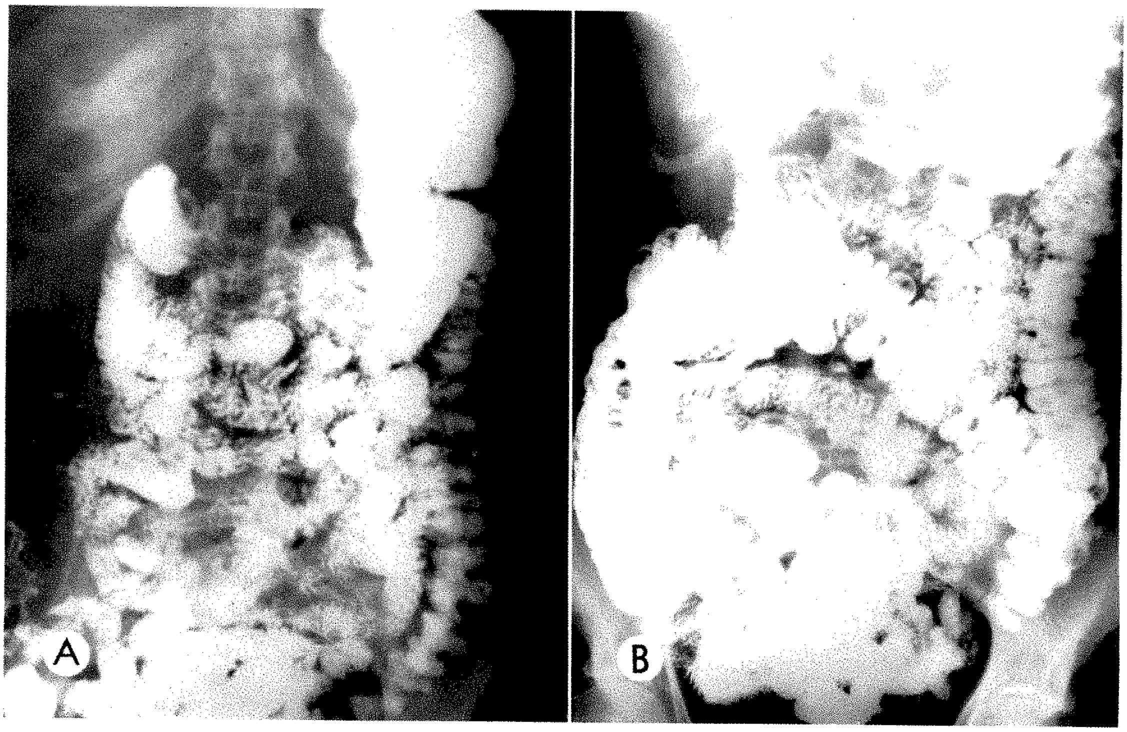


Fig. 5. Case 8. (*A*) Posteroanterior prone and (*B*) anteroposterior supine roentgenograms of the abdomen with barium outlining the stomach, small intestine and colon show scattered diverticula of varying sizes throughout the entire small bowel.

in patients after bowel sterilization with broad-spectrum antibiotics lends strong support to this theory.

The steatorrhea associated with intestinal diverticulosis usually occurs in the elderly patient, and is often accompanied by symptoms of partial intestinal obstruction. The onset is usually abrupt and heralded by the sudden onset of diarrhea. The defect of fat absorption is relatively mild, and any other absorptive errors, if present, are also mild.

Small bowel roentgen examination generally does not demonstrate the so-called deficiency or disordered motor function pattern. The administration of broad-spectrum antibiotics will usually abolish this steatorrhea; however, relapses are frequent and control often is difficult. If neoplastic involvement of the pancreas or bowel can be excluded in a patient initially presenting with steatorrhea late in life, diverticulosis of the small intestine should always be a prime consideration.

There are certain similarities between patients with macrocytic anemia associated with intestinal diverticulosis and Addisonian pernicious anemia. In the former group the patient may present clinically with glossitis, peripheral neuropathy, icterus and subacute combined degeneration of the cord. The peripheral blood and marrow picture as well as the Schilling test may be indistinguishable in the two types. The patient with diverticulosis will often manifest concomitant steatorrhea as well. The impaired absorption of vitamin B_{12} is improved by the parenteral administration of liver extract and vitamin B_{12} in both entities. However, free hydrochloric acid usually can be demonstrated in the gastric secretion of patients with diverticulosis but not in the pernicious anemia patient (except rarely). Additionally, broad-spectrum antibiotic therapy will beneficially alter the absorption defect only in the patient with diverticulosis. This is the only instance of macrocytic anemia showing such

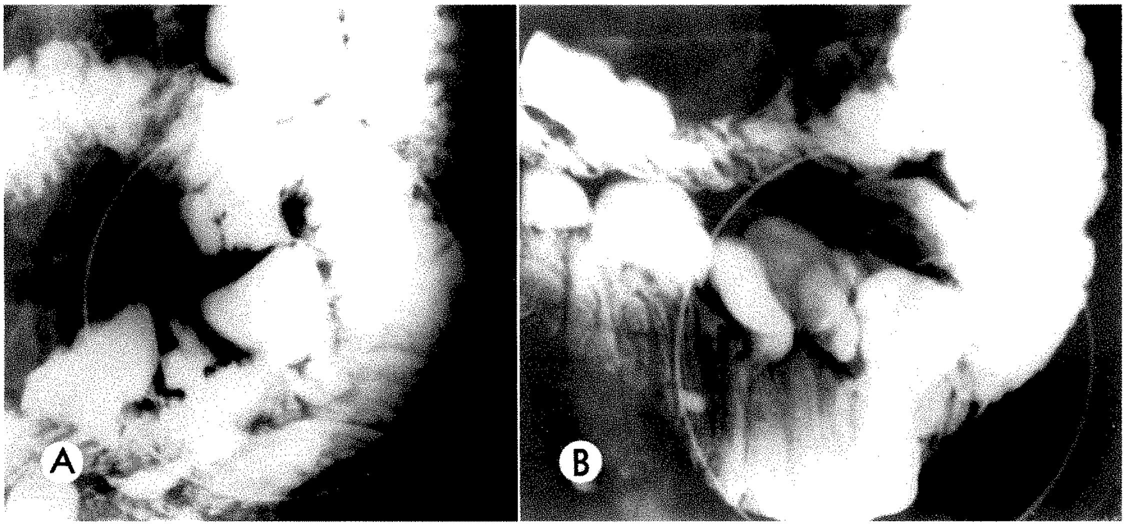


FIG. 6. Case 9. (*A* and *B*) Compression "spot" roentgenograms at the time of small intestinal examination with barium demonstrate multiple wide-necked diverticula arising from the proximal jejunum and appearing as smooth, rounded densities with no evident mucosal pattern.

a consistent type of improvement with antibiotics.

ROENTGEN DIAGNOSIS

Plain roentgenograms of the abdomen are usually unremarkable in the asymptomatic unsuspected case. Air fluid levels in the erect or horizontal beam position, however, may closely resemble the roentgen appearance of intestinal obstruction. Rarely, will a diverticulum attain such size as to produce a soft tissue mass with displacement of bowel loops. Enteroliths may be observed, but it is usually impossible to determine whether they lie within the diverticulum or the bowel lumen.

The diverticula demonstrated by barium meal examination are smooth hemispheric densities with no evident mucosal pattern, and are situated on the mesenteric border, varying in size from less than a centimeter to several centimeters in greatest diameter. They are usually multiple, with wide necks, and occur with decreasing frequency distalward from the ligament of Treitz. Diverticula of the ileum are uncommon, but when encountered, are usually quite small and situated in the terminal portion as contrasted with a Meckel's type of diverticulum. In the erect position, gas-fluid levels are observed with the barium settling to the most dependent position. An associated disordered small bowel pattern is rarely

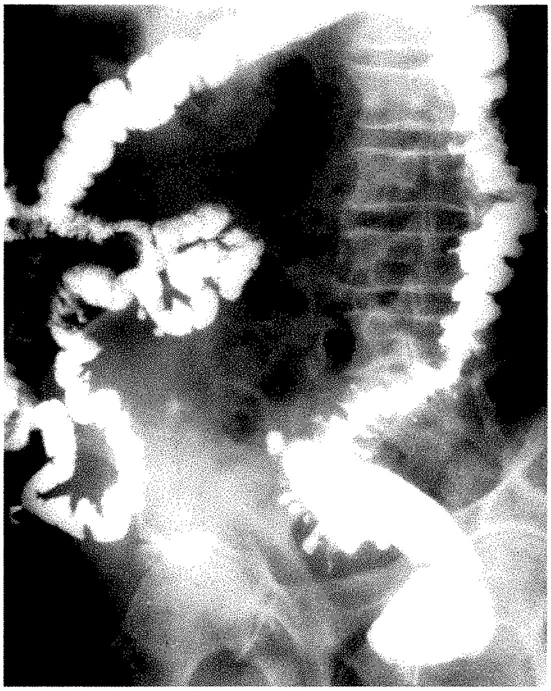


FIG. 7. Case 10. Oblique barium enema study of the abdomen shows several small diverticula in the terminal portion of the ileum into which barium has refluxed. There is associated diverticulosis involving the descending colon and rectosigmoid.

seen, and the barium column usually proceeds in a normal fashion. Residual opaque densities may be demonstrated on delayed roentgenograms but the diverticula, for the most part, empty readily.

THERAPY

In brief, the therapeutic approach to jejunal diverticulosis is primarily dependent on the clinical history. If the symptoms are relatively mild, conservative management is recommended in an attempt to avoid over-distention, constipation and increased intra-intestinal pressure. Surgical intervention is the only corrective measure available, and is necessitated in those cases with prolonged, intractable symptoms, or those presenting with an acute intra-abdominal complication. Segmental resection with end-to-end entero-enterostomy, when feasible, is the procedure of choice. The syndrome of macrocytic anemia and/or steatorrhea will respond initially to broad-spectrum antibiotic therapy. Surgical excision is effective in correcting the absorptive defects, and this mode of therapy should be utilized when medical therapy fails.

CLINICAL MATERIAL

We have arbitrarily included in our series all cases which consisted of more than 3 small intestinal diverticula, arising distal to the ligament of Treitz and involving any portion of the small intestine to the region of the terminal ileum. Employing these criteria, we have been able to collect 12 such cases and have tabulated the clinical manifestations in Table 1. One of these cases (Case 12) presented with chronic low grade intestinal obstruction, eventually necessitating surgical intervention. We are reporting this case in greater detail although it is included in our tabular compilation.

There were 6 male and 6 female patients, ranging in age from 42 to 83 years. The association of colonic and duodenal diverticula was encountered respectively in 6 patients; 2 of these patients had diverticula of the colon, duodenum and small intestine. The maximum size of the diverticula ranged from 0.5 to 8 cm. in greatest diameter, the majority measuring about 4 cm. Three to 5 diverticula were encountered in 7 patients. In the 5 remaining patients, the diverticula were too numerous to count (Table 1).

The commonest clinical symptom was postprandial abdominal pain, which was noted by 6 of the 12 patients. Diarrhea and eructation were present in 4 cases and nausea, vomiting and anorexia in 3. Melena was a presenting symptom in 1 patient, with no gastrointestinal abnormalities other than the small bowel diverticulosis being found. After conservative therapy, the bleeding ceased.

Associated gastrointestinal disorders included hiatal hernia in 3 cases and chronic duodenal ulcer disease in 2 others. Two patients gave a history of choledocholithiasis.

Laboratory findings were essentially noncontributory and no patient in our series showed evidence of an absorptive defect.

Figures 1 through 7 are representative roentgenograms of patients with small bowel diverticula in our series.

REPORT OF A CASE

CASE 12. L.M., a 52 year old Negro female, was admitted to Montefiore Hospital in April, 1959, complaining of intermittent dull left lower quadrant pain, a gradual abdominal enlargement and a bloating sensation after meals even with a small amount of food intake. Nine weeks prior to admission, she began to experience diarrhea which increased in frequency to 10–12 small watery bowel movements daily. There was no blood, pus or mucus per rectum. She had lost 7 pounds in weight during this period.

Physical examination revealed a thin female with a distended, tympanitic, nontender abdomen. No masses could be palpated and no fluid wave or shifting dullness was demonstrable. Urinalysis, hemogram and blood chemistries were normal. Stool specimens were negative for blood, ova or parasites. Serum carotene level, Schilling and radioactive triolein

Table I

SUMMARY OF CASES

Case No.	Patient	Age	Sex	No. of Diverticula	Post-prandial Abdominal Pain	Nausea, Vomiting and/or Anorexia	Eructation	Diarrhea	Bleeding	Associated Lesions
1 (Fig 1)	Z.R.	83	M	5	+					Chronic duodenal ulcer; hiatal hernia; duodenal diverticula
2	R.P.	61	F	3-4	+		+			Hiatal hernia
3 (Fig 2, *A* and *B*)	A.S.	61	M	3-5	+					Healed myocardial infarct; common duct stone with icterus; colon diverticula; duodenal diverticula
4 (Fig. 3, *A–C*)	C.D.	72	F	Numerous	+	+		+		
5	H.K.	72	M	Numerous	+					Myocardial infarct; arteriosclerotic cardiovascular disease; benign prostatic hypertrophy; duodenal diverticula
6 (Fig. 4, *A* and *B*)	A.P.	81	M	Numerous		+				Carcinoma of the pancreas with cholangitis; colon diverticula
7	J.A.	73	F	Numerous		+				Carcinoma of the right upper lobe; colon diverticula
8 (Fig. 5, *A* and *B*)	C.M.	42	F	Numerous					+	
9 (Fig. 6, *A* and *B*)	S.J.	65	M	Numerous	+			+		Hiatal hernia; colon diverticula
10 (Fig. 7)	A.M.	78	M	5				+		Chronic duodenal ulcer; chronic gallbladder disease with common duct stones; colon diverticula
11	A.L.	69	F	4			+			Arteriosclerotic cardiovascular disease; congestive heart failure; duodenal diverticula
12 (Fig. 8, *A–D;* and 9)	L.M.	52	F	Numerous	+	+	+	+		Colon diverticula; duodenal diverticula

tests all were within normal limits, and there was no evidence of an absorptive defect. Bone marrow aspiration was interpreted as showing normoblastic erythroid hyperplasia.

Upper gastrointestinal and small bowel examination revealed numerous diverticula varying in size from less than 1 cm. to about 5 cm. These extended from the proximal jejunum, just distal to the ligament of Treitz, to the region of the junction of the jejunum and ileum (Fig. 8, *A–D*).

The patient's hospital course was that of low grade partial intestinal obstruction, characterized by abdominal distention, borborygmi, food intolerance and progressive weight loss. She was treated conservatively with antispasmotics and mild sedation, but her general condition progressively deteriorated so that she was barely able to ingest even liquids. Because she remained intractable to medical therapy, a laparotomy was performed.

On surgical exploration multiple diverticula were encountered, involving approximately 105 cm. of small bowel beginning just distal to the ligament of Treitz. The involved bowel was dilated and hypertrophied. Because the distal small bowel and colon appeared uninvolved, a segmental resection with end-to-end anastomosis was performed.

Microscopic sections of the diverticula

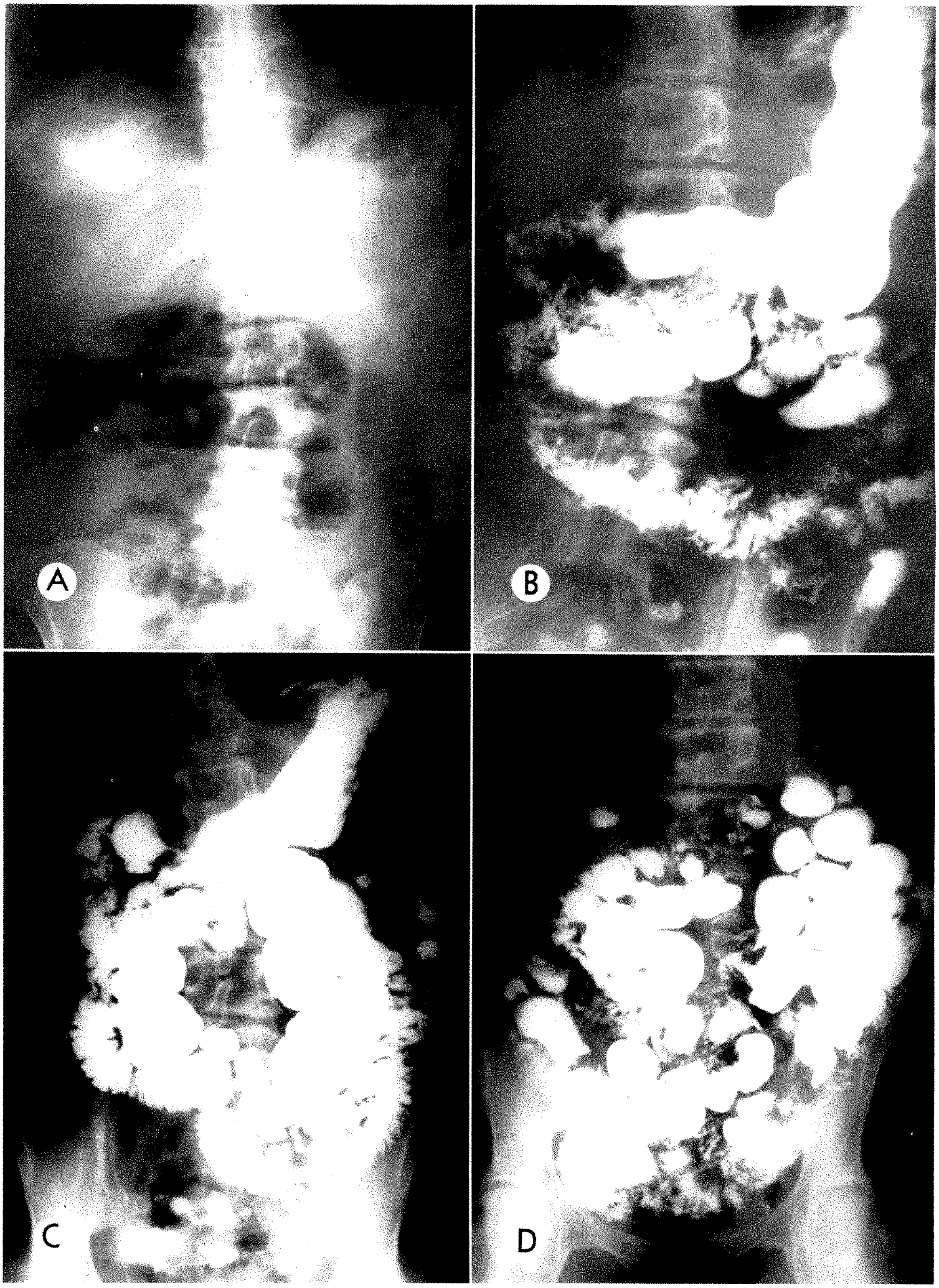


FIG. 8. Case 12. (*A*) Posteroanterior erect plain roentgenogram of the abdomen shows, in addition to several dilated small bowel loops, numerous oval radiolucent zones containing fluid levels with no evident mucosal

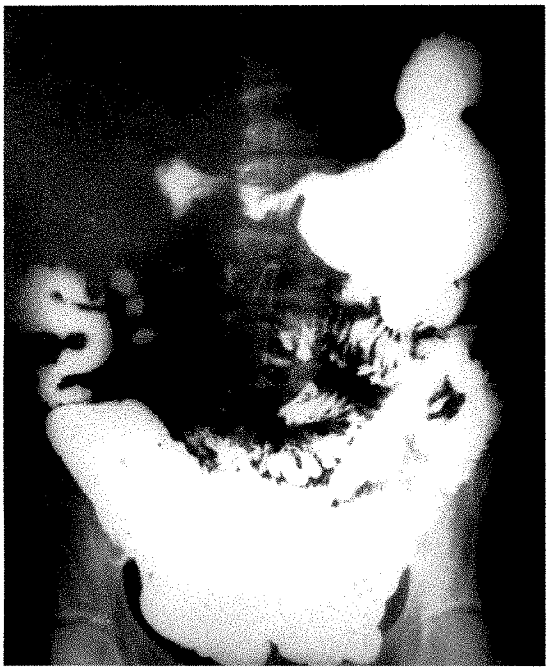


Fig. 9. Case 12. Posteroanterior prone roentgenogram of the abdomen 3 years after surgical resection of jejunal diverticula still demonstrates scattered jejunal diverticula, smaller and less numerous when compared with Figure 8.

demonstrated the presence of the muscularis mucosae surrounding the mucosa. However, the external muscular layers were markedly thinned and represented by only a few small strands; a moderate round cell infiltration was present.

Postoperative recovery was uneventful and the patient remained asymptomatic for 3 years when she was re-admitted with nausea, vomiting and periumbilical pain. Gastrointestinal examination at this time revealed duodenal ulcer disease and 10–15 duodenal and jejunal diverticula, much smaller than those demonstrated prior to the previous surgical procedure (Fig. 9). Her present symptoms responded to a medical ulcer regimen, and she was discharged to her home.

SUMMARY AND CONCLUSION

1. The literature dealing with small bowel diverticula (exclusive of duodenal diverticula) is reviewed.
2. The incidence, types, pathogenesis, clinical manifestations, complications, roentgen diagnosis and management of this entity are discussed.
3. A tabular summary of the findings in our personally observed series of 12 cases with small bowel diverticulosis is presented.
4. The roentgen findings in selected cases are illustrated.
5. One patient in our series presenting with chronic low grade intestinal obstruction requiring surgical intervention is discussed in some detail.

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pattern. (*B* and *C*) Posteroanterior prone roentgenograms of the abdomen with barium in the stomach and small bowel outlining numerous large diverticula of the jejunum and proximal ileum. (*D*) Posteroanterior prone abdominal roentgenogram again shows barium in numerous small intestinal diverticula.

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Vol. 92, No. 5

INTRAMURAL INTESTINAL HEMORRHAGE*

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INTRAMURAL intestinal hemorrhage as a result of trauma has been well documented. With the increasing use of anticoagulants, bleeding of a similar type in the absence of any specific trauma is becoming more frequent. Intramural hemorrhage may also occur spontaneously in many conditions with a bleeding diathesis such as hemophilia, Henoch's purpura, idiopathic thrombocytopenic purpura, leukemia, etc. Experience with several of these diseases has provided an opportunity to analyze the roentgen changes of these conditions. Recognition of intramural hemorrhage in intestinal bleeding may be critical since it has a significant bearing on therapy and prognosis.

HISTORY

Trauma as a cause of intramural intestinal hematomas has been noted since 1838 and the pathologic features either at autopsy or at surgical exploration have been described.[25,34] The first roentgen demonstration was reported in 1948 in a patient who developed ileus following an abdominal injury.[21] Subsequently, many case reports have appeared in the literature. The roentgen criteria described by Felson and Levin[11] are thickening and spreading of the valvulae conniventes and an intramural mass simulating a coiled-spring appearance.

With the initiation of anticoagulant therapy, bleeding complications were observed.[1,4,14,20,24,33] Berman and Mainella[5] in 1952 reported hemorrhage into the wall of the small and large bowel, peritoneum and retroperitoneal tissues in 5 cases receiving anticoagulants. In none of these cases were roentgen examinations performed and the diagnoses were confirmed at surgical exploration or at autopsy. In 1958, Pearson and MacKenzie[28] reported a case of hemorrhage into the wall of the jejunum. Although contrast studies were not done, air outlining the upper jejunal loops indicated that the folds were irregular and the lumen narrowed. Culver *et al.*[7] in 1961 described a case of jejunal intraumural hematoma on roentgen examination of the small bowel in a patient on anticoagulant therapy. Shortly thereafter, barium meal studies in 2 cases involving the duodenum and 3 the jejunum were reported.[31,39] Additional cases have since been recognized and documented.[32]

Gastrointestinal hematomas are a bizarre complication of hemophilia and few such cases have been recorded radiologically.[38] As is the case with anticoagulant therapy, bleeding in hemophilia may occur in the bowel wall and mesentery and the patient may clinically present with abdominal pain, melena, abdominal mass or intestinal obstruction. Any portion of the gastrointestinal tract may be involved but roentgenographic documentation has been reported in the small intestine and colon only.[19,40]

Henoch-Schönlein's anaphylactoid purpura is frequently associated with gastrointestinal complaints. The cutaneous manifestations were noted by Willan[37] in 1808, and joint manifestations and purpura by Schönlein in 1837. Not until 1874 were the gastrointestinal manifestations pointed out by Henoch.[16] The disease affects young men predominantly. Involvement of both the large and small intestine as well as the stomach has been described.[8,15,36] Surgical intervention in one patient explored for acute abdominal pain of unknown etiology revealed the appearance of acute regional enteritis.[8] Purpuric skin lesions appeared the day following laparotomy. Wound dehiscence 9 days later permitted a second look at the ileum which at that time had returned to normal. Gastrointestinal symptoms frequently precede the appearance of petechiae. However, it is not until petechiae appear that the diagnosis becomes apparent.[2,6,22,27]

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In periarteritis nodosa, despite the frequent occurrence of pathologically demonstrated lesions in the gastrointestinal tract, alterations gross enough to be seen roentgenologically are rare. Abdominal pain has been noted in 50 to 70 per cent of the cases and gross intestinal lesions are seen at autopsy in 20 to 40 per cent. Intestinal changes consist of mucosal hemorrhages and ulcerations which may be solitary and minute or confluent, depending on the site and extent of vascular involvement.[13,17] Submucosal edema and mucosal ulcerations present the roentgen appearance of ulcerative jejunitis.[9] When the mucosa is involved, changes may be seen on sigmoidoscopy which simulate ulcerative colitis.[10] Bleeding into the bowel wall may occur associated with neoplasms such as leukemia, myeloma, lymphomas or metastatic carcinomas. The explanation for this bleeding diathesis in these cases is not always clear. Thrombocytopenia, hypofibrinogenemia, circulating anticoagulants, activation of the fibrinolysin system and dysproteinemias have been implicated.[23,26,29,30,35]

ROENTGEN DIAGNOSIS

A preliminary roentgenogram of the abdomen may demonstrate significant findings. If there is a solitary hematoma of sufficient size, it may be recognized either by its increased density or the displacement of loops of the small or large bowel. If the hemorrhage is confined to the bowel wall, varying degrees of paralytic ileus, narrowing of the bowel lumen or localized filling defects producing marked scalloping of the bowel may be seen. Differentiation from infarction on the preliminary roentgenogram alone can be difficult.

With barium studies, the roentgen changes due to intramural hemorrhage in all these conditions have many features in common. The variation in the roentgen appearance seems to be a reflection of the differences in the extent of intramural hemorrhage, the presence of mucosal edema and of intestinal secretions. Changes in the small intestine have been reported more frequently than in the large intestine and involvement of the esophagus and stomach has not been documented. The roentgen changes are usually segmental but may be diffuse. The involved segments of small intestine reveal varying degrees of rigidity and uncoiling of the loops, depending on the degree and extent of bleeding into the bowel wall and mesentery. Marked rigidity and separation of the loops of bowel have not been observed except in one patient with hemorrhage secondary to prostatic carcinoma with metastases (Fig. 1*A*). In this case the bleeding was so profuse that it produced marked rigidity and narrowing of a long segment of the small bowel (Fig. 1*B*). As a rule mechanical or clinical obstruction is not prominent although some degree of narrowing of the bowel lumen is frequent. Proximal to the lesion, transient intussusception was noted in one case (Fig. 2*A*). When bleeding ceases, resorption of the hemorrhage and complete resolution appear to be the rule (Fig. 2*B*).

The small intestine fold pattern may have a highly characteristic appearance and varies slightly with the different entities producing intramural hemorrhage. There is a uniform, regular thickening of the folds, sharp delineation of their margins, and a parallel arrangement producing a symmetric spike-like configuration simulating a stack of coins (Fig. 2*A*; and 3*A*). This stacked coin appearance is the most frequent and prominent alteration and is more striking in the jejunum than distally because of the normally greater prominence of the jejunal folds. There is a conspicuous lack of spasm or changeability of folds.

In idiopathic thrombocytopenic purpura and Henoch's purpura, in addition to hemorrhage, varying degrees of mucosal edema can occur with the accumulation of

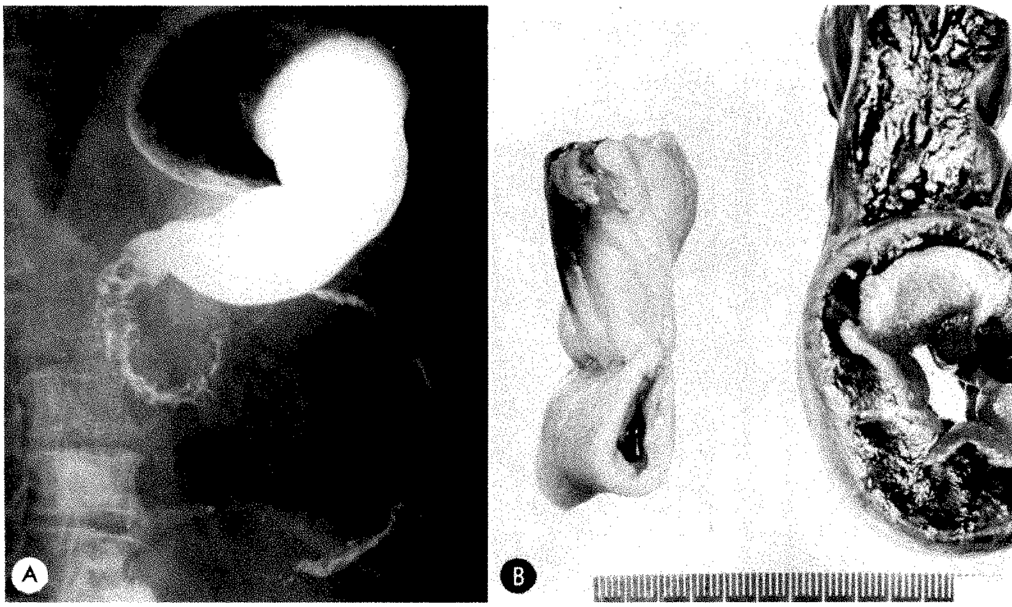

FIG. 1. (*A*) Massive hemorrhage in a case of prostatic carcinoma. At 3 hours barium study outlines only the duodenum and proximal jejunum. In the duodenum, the lumen is narrow and the folds are regularly thickened. The jejunum is markedly narrowed and the folds are obliterated. There is marked rigidity and C-shaped configuration secondary to intramural hemorrhage. (*B*) Gross section of the involved segment of the jejunum and mesentery revealing marked intramural bleeding with elevation of the mucosa and considerable narrowing of the lumen. Hemorrhage into the mesentery is also identified.

fluid in the lumen of the bowel. The increased secretions and edema may blur the sharpness and symmetry of the folds. Despite this, the folds are regularly arranged and have a constant appearance (Fig. 4*A*; and 5). In the more severe cases, this increased secretion and edema produce effacement of the folds and the barium column may become segmented and fragmented (Fig. 6, *A* and *B*). Occasionally, scalloping and thumbprinting due to localized hemorrhage or edema is seen.

Bleeding into the mesentery is a frequent finding, especially in patients with anticoagulants and in hemophiliacs. Evidence of a mass with flattening of the folds on the mesenteric side of the bowel and separation and uncoiling of bowel loops is noted (Fig. 7*A*). If the mesenteric hemorrhage is large, a hammock-like configuration of the bowel segment may result (Fig. 2*A*; and 3, *A* and *B*).

Intramural hemorrhage involving the colon produces two types of changes which may merge imperceptibly with one another. It may produce changes similar to the small bowel reflected in the thickening and spreading of the mucosal folds. In other cases, it may produce a mass simulating a tumor. When the folds only are involved, they appear thickened, blunted, and have a regular course with no evidence of ulceration (Fig. 8; and 9*A*). With more extensive bleeding, smooth scalloped defects (thumbprinting) along the contours of the bowel lumen may be noted (Fig. 10, *A* and *B*; and 11). When the bleeding is segmental, the findings may be those of a submucosal mass with local narrowing of the lumen, varying degrees of stretching of the mucosa which may be completely effaced, sloping margins and no evidence of significant obstruction to the flow of barium (Fig. 12*A*). Associated bleeding into the mesocolon and into the mesentery of the small bowel produces evidence of an extrinsic mass along the mesenteric border of the bowel (Fig. 9*A*).

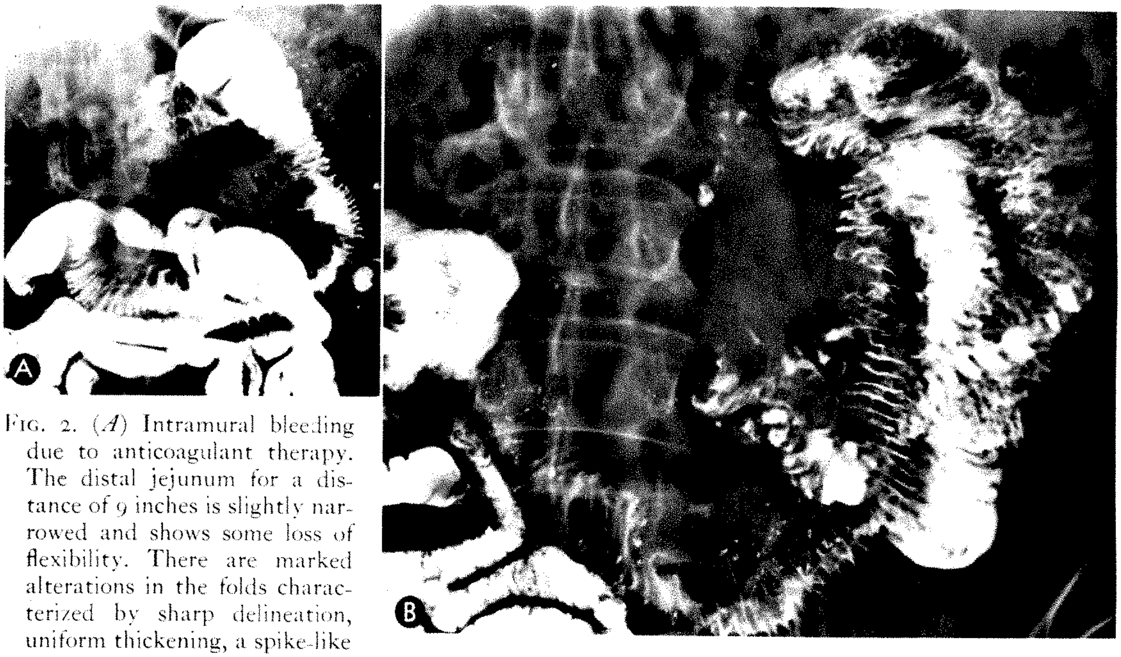

FIG. 2. (*A*) Intramural bleeding due to anticoagulant therapy. The distal jejunum for a distance of 9 inches is slightly narrowed and shows some loss of flexibility. There are marked alterations in the folds characterized by sharp delineation, uniform thickening, a spike-like configuration, and symmetric and regular spacing simulating a stack of coins. There is no mucosal ulceration or nodularity suggestive of inflammatory or neoplastic process. The bowel immediately proximal to this segment is slightly dilated with transient intussusception. The distal bowel reveals varying degrees of dilatation. The remaining small intestine is normal. (*B*) Two days later there has been considerable resolution in the degree and extent of involvement. The folds are less thickened and the bowel lumen has become wider. Autopsy 9 months later showed no abnormality of the small bowel.

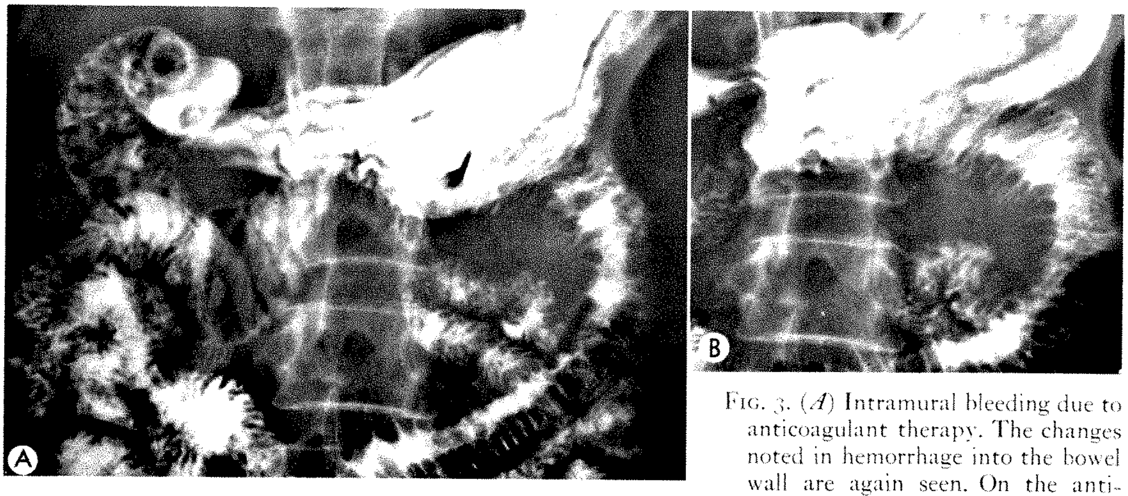

FIG. 3. (*A*) Intramural bleeding due to anticoagulant therapy. The changes noted in hemorrhage into the bowel wall are again seen. On the antimesenteric side, the regularly thickened folds have a striking uniform appearance. On the opposite mesenteric side, the folds are irregularly flattened, not only by the intramural hematoma but by blood in the mesentery as well. (*B*) Extrinsic pressure by the mesenteric hematoma is prominent.

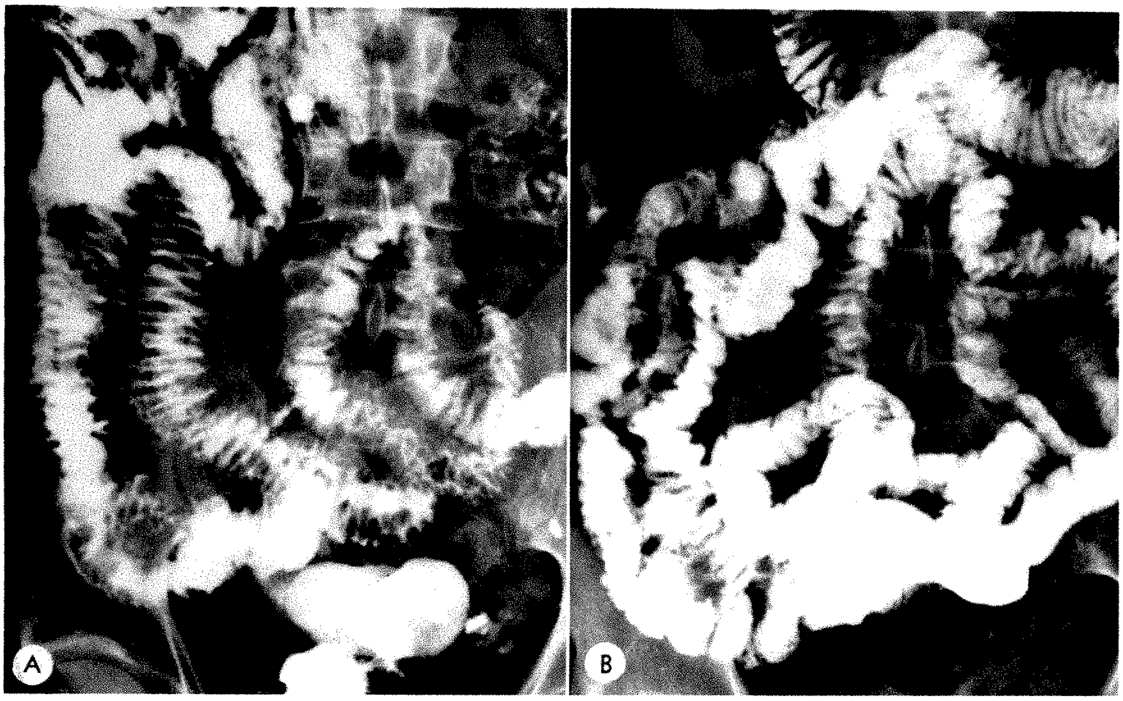

Fig. 4. (*A*) Idiopathic thrombocytopenic purpura with hemorrhage into the small intestinal wall. The folds in the distal ileum are thickened and have a stacked coin appearance. The appearance is similar to that noted in intramural bleeding incident to anticoagulant therapy. The findings were confirmed at surgery during splenectomy. (*B*) Five weeks later, the abnormal findings have completely resolved.

Follow-up examinations of these patients have revealed resolution of the roentgen findings in a short period of time (Fig. 4*B*; 7*B*; 9*B*; and 12*B*).

DIFFERENTIAL DIAGNOSIS

The roentgen alterations combined with the clinical history are usually sufficiently characteristic to warrant a definitive diagnosis. The one exception and perhaps the most important in view of therapy is differentiation of the roentgen changes due to mesenteric vascular occlusion and those due to bleeding from anticoagulant therapy. The roentgen alterations in both of these conditions may, at a glance, appear similar. Since clinically a patient who is on anticoagulants may also develop a mesenteric vascular occlusion, the clinical situa-

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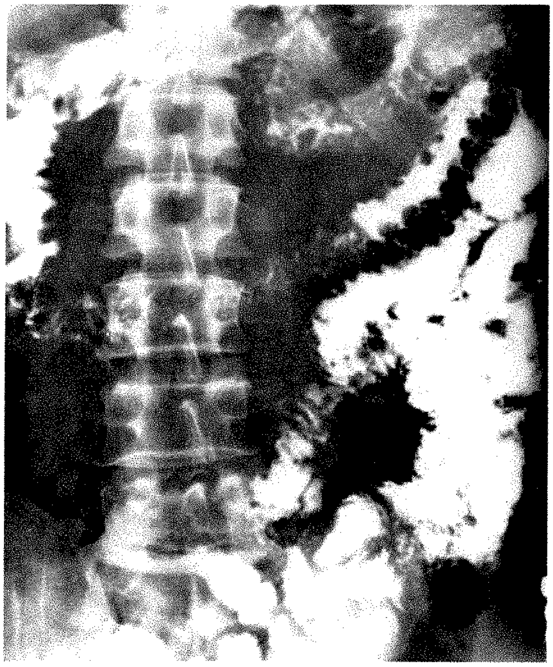


Fig. 5. Henoch's purpura. Almost the entire small intestine is involved. The changes are characterized by irregular thickening of the mucosal folds, increased secretions and minimal segmentation distally. The bowel lumen is of normal caliber.

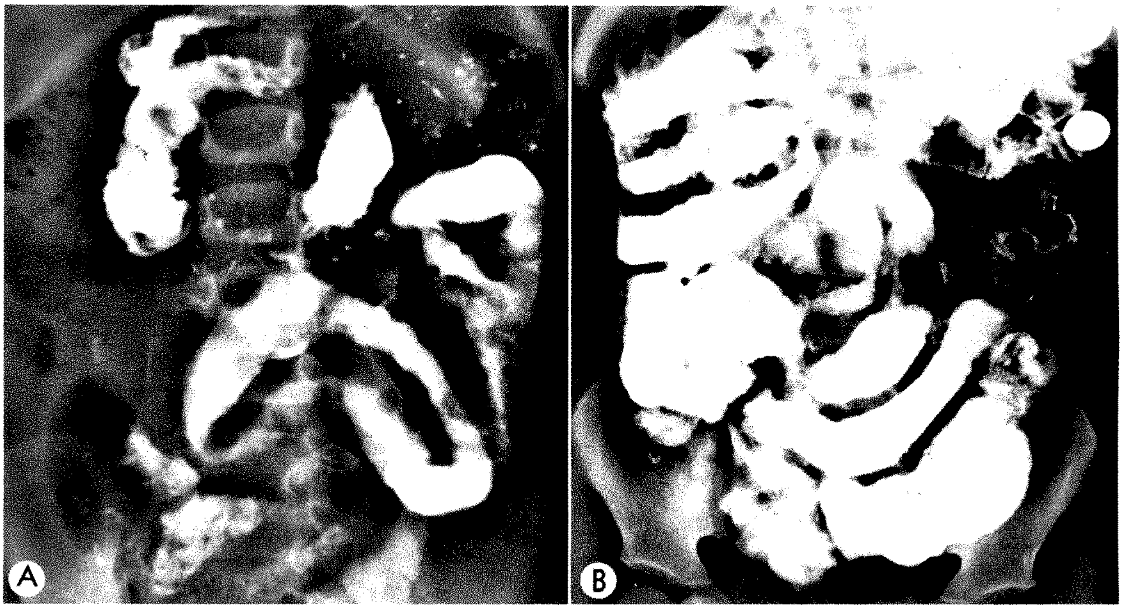
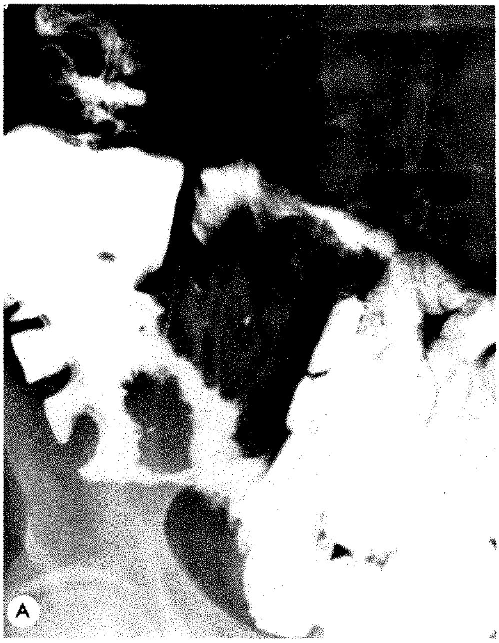


Fig. 6. (*A*) Henoch's purpura. The small intestine is slightly dilated. The mucosal pattern is coarse and in some areas effaced, presumably due to increased secretions and mucosal edema. (*B*) Three days later, the small bowel changes are less marked.



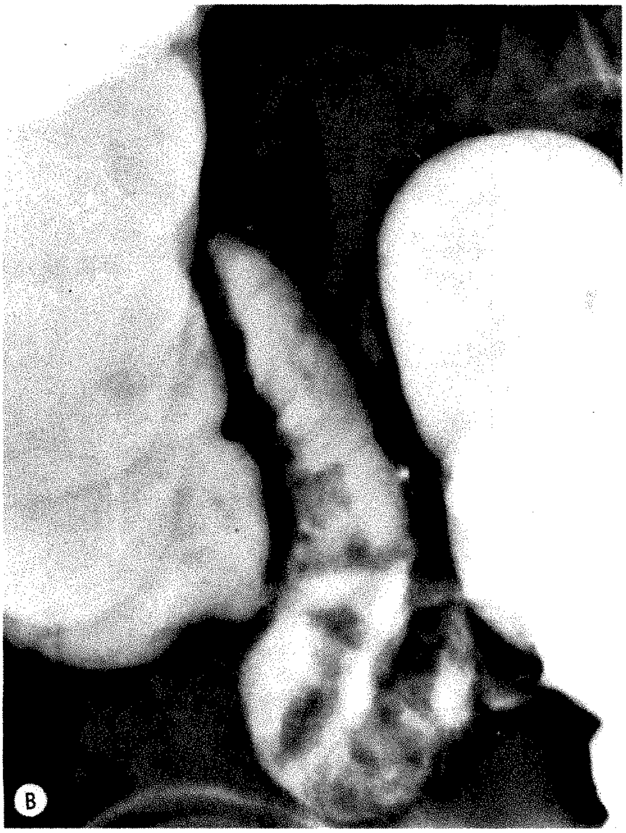


Fig. 7. (*A*) Hemophilia. The terminal ileum is moderately rigid with some narrowing of the lumen and irregularity of the contours. The folds are thickened with no ulceration, nodularity or infiltration. There is a mass, the size of an orange, displacing the small bowel cephalad and stretching the caput coli. There is no evidence of obstruction, spasm or irritability. (*B*) Two months later, there is complete resolution of the changes.

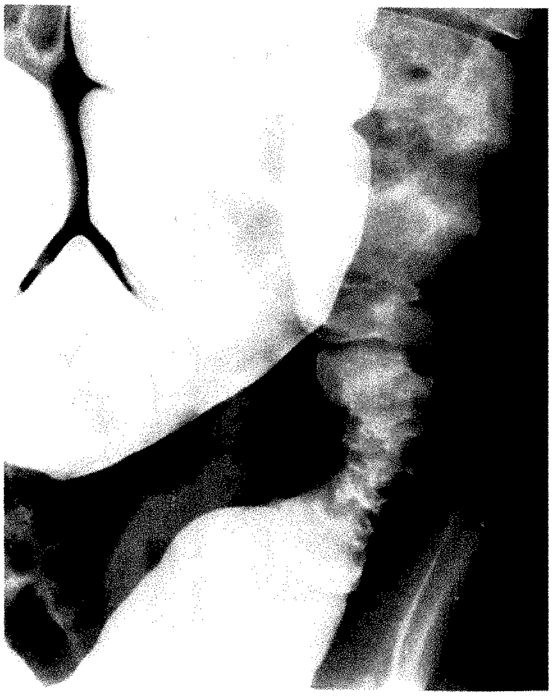


Fig. 8. Hemophilia with bleeding into the colonic wall (examination performed 3 days after onset of bleeding). Minimal changes in a segment of the mid-descending colon are manifested by slight eccentric narrowing of the lumen, exaggerated folds and evidence of a mass medially. No diverticula are seen. The findings simulate the resolving stage of acute diverticulitis.

tion may not permit certain differentiation. In favor of anticoagulant changes, however, is the stacked coin appearance, stability of the fold pattern, lack of spasm or irritability, presence of a mesenteric mass and complete resolution of the roentgen findings. In mesenteric vascular occlusion, on the other hand, a localized mesenteric mass due to bleeding is uncommon. The folds exhibit varying degrees of edema, spasm and irritability (Fig. 13*A*). The usual sequel is a stricture at the site of the maximally infarcted segment (Fig. 13*B*). In some cases, however, differential diagnosis is impossible since complete resolution of all changes has also been noted in mesenteric vascular occlusion (Fig. 14, *A* and *B*).

Inflammatory lesions of the small bowel

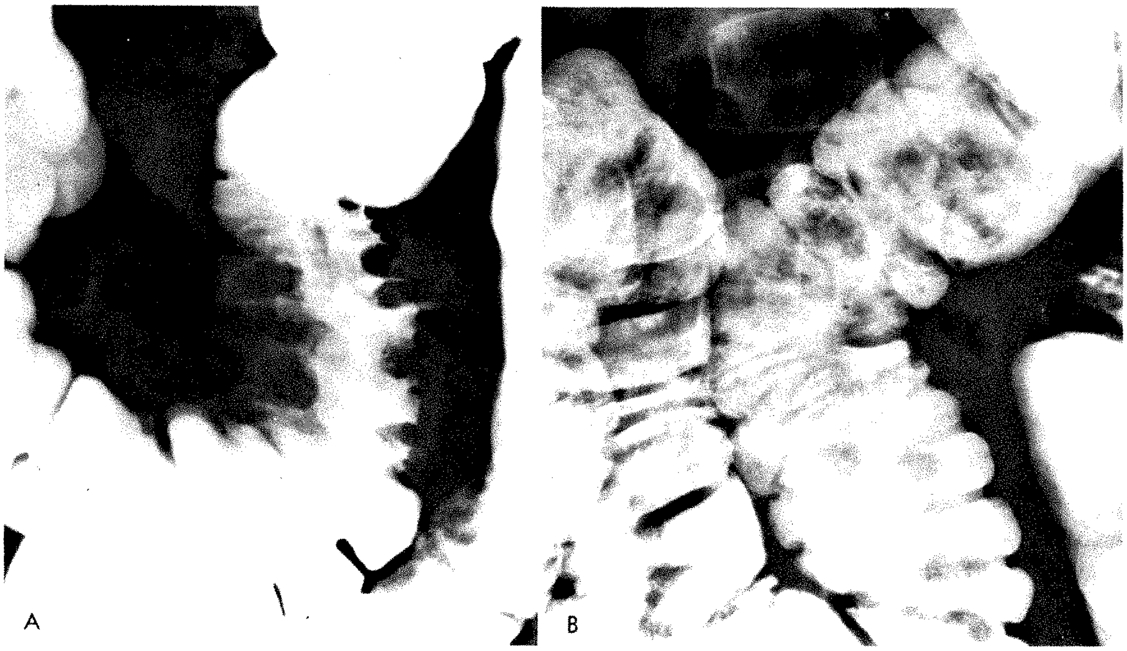


Fig. 9. (*A*) Hemophilia. A short segment of transverse colon is slightly narrowed and the contours are regularly fluted. The changes are perhaps slightly more marked on the superior aspect at the attachment of the mesocolon, indicating possible hemorrhage in this region. (*B*) Ten weeks later, the colon has returned to normal.

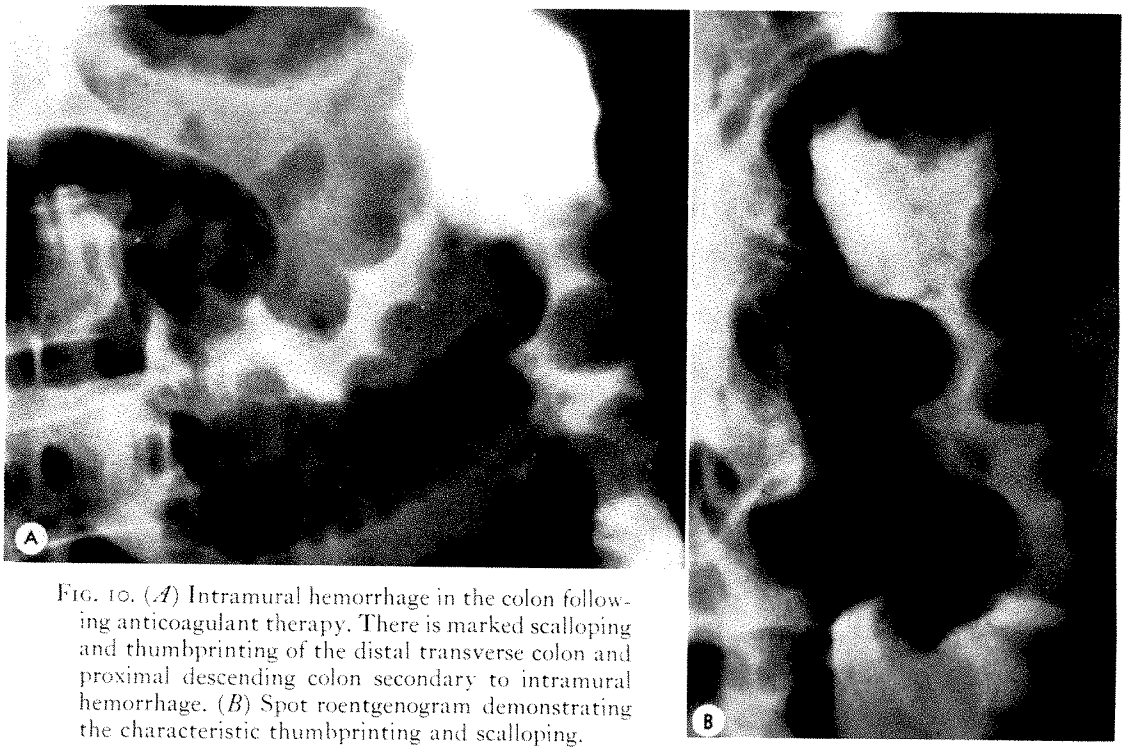


Fig. 10. (*A*) Intramural hemorrhage in the colon following anticoagulant therapy. There is marked scalloping and thumbprinting of the distal transverse colon and proximal descending colon secondary to intramural hemorrhage. (*B*) Spot roentgenogram demonstrating the characteristic thumbprinting and scalloping.

can usually be differentiated from intramural hemorrhage. In the early stages there is marked spasm and irritability while later ulceration, inflammatory polyps, marked rigidity and separation of the loops of bowel are evident. Neoplastic diseases of the small bowel, specifically lymphosarcoma, frequently reveal ulceration, localized dilatation, tumor nodules or multiple areas of involvement.

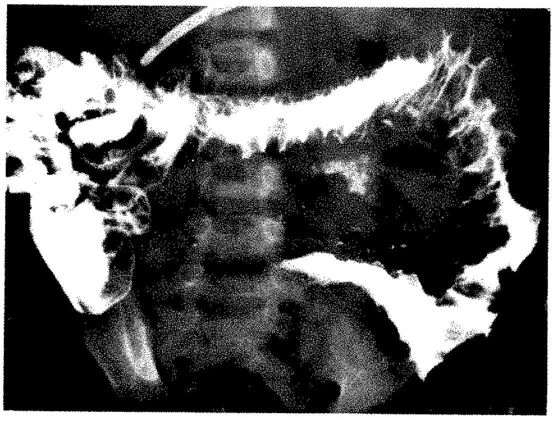


Fig. 11. Massive rectal bleeding in a 3 year old female with biliary duct atresia. Delayed roentgenogram in a gastrointestinal series reveals symmetric and markedly exaggerated mucosal folds in the transverse and descending colon secondary to intramural hemorrhage. In addition, thumbprinting or scalloping in the region of the splenic flexure is evident. Tumor nodules, ulceration or a mesenteric mass are not seen.

Clinically, the malabsorption syndrome and cases of exudative enteropathy are not a problem in differential diagnosis. On occasion, there may be certain similarities in the roentgen picture. Sprue is a diffuse process associated with pliability of the folds, stretching of the valvulae conniventes and dilatation. There is practically always evidence of segmentation. Edema of the folds of the colon has been noted in Whipple's disease, nephrosis, exudative enteropathy, cardiac failure, hypo-albuminemia, and in cirrhosis of the liver. This edema most often is diffuse. On occasion it is marked, segmental and produces scalloping of the bowel similar to intramural hemorrhage. The clinical history, however, is usually characteristic.

SUMMARY

Intramural hemorrhage of the small bowel and colon as a complication of hem-

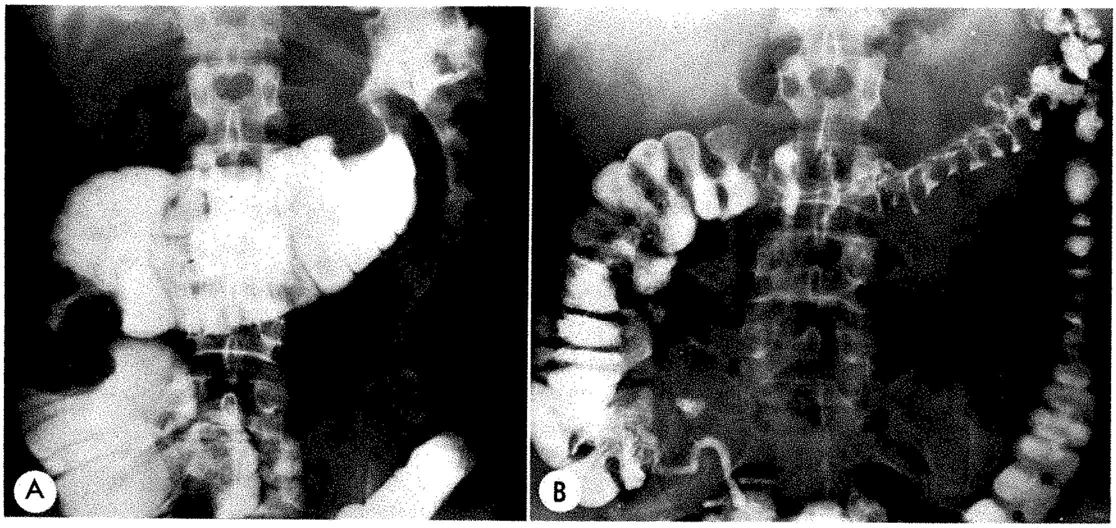


FIG. 12 (*A*) Henoch's purpura involving the colon. Two segmental areas of narrowing are characterized by sharply defined margins, a concentric lumen which retains some degree of flexibility, and irregular thickening of the folds without mucosal ulceration. Barium flowed readily through the narrowed segments. The appearance is consistent with a circumferential submucosal lesion. (*B*) Ten weeks later, the colon appears normal.

orrhagic diathesis and anticoagulant therapy is being recognized with increasing frequency. Roentgen study has revealed a characteristic pattern in many of these cases. Recognition of this pattern and differentiation from other discrete lesions of the intestinal tract are important since operation is unnecessary and resolution is the rule. The roentgen changes were studied in a variety of patients with hemorrhagic diathesis and in patients bleeding from anticoagulant therapy. The differential diagnosis from mesenteric vascular occlusion and other disorders is described.

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The authors would like to thank Dr. Joseph Dunne and Dr. Frank Matus of East Orange Veterans Hospital and Dr. Isidore Katz of State University of New York for the use of

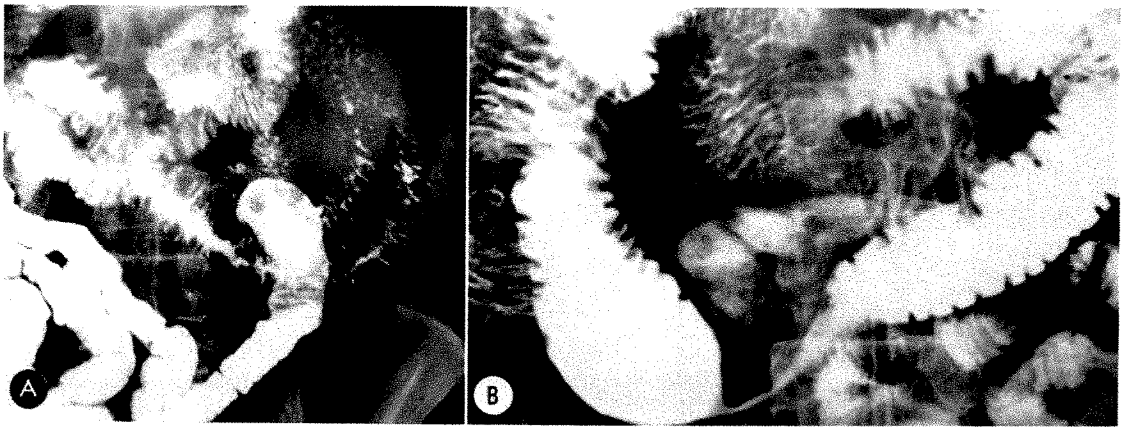

FIG. 13 (*A*) Segmental infarction. There is marked spasm and irritability of the mid-jejunum. The involved loops are uncoiled. The folds are thickened and irregular and have a fragmented appearance. (*B*) Two months later, stricture formation has resulted, a frequent sequel in this condition.

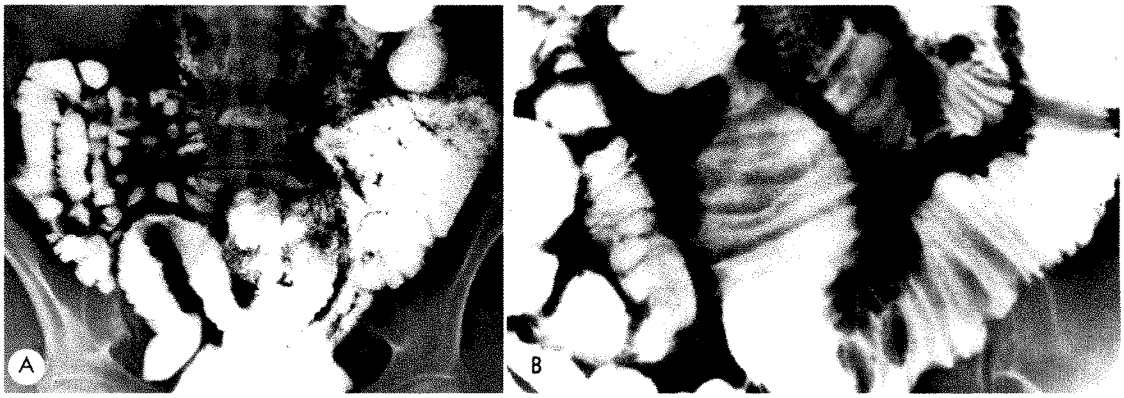

Fig. 14. (*A*) A 58 year old woman, 3 months following myocardial infarction and on anticoagulation therapy developed mid-abdominal pain. A small intestinal study revealed an abnormal ileal loop characterized by abnormal motor activity, thickening and flattening of the folds without ulceration or nodularity. (*B*) Spot roentgenogram. Roentgenologically, it was not possible to differentiate with certainty between hemorrhage due to anticoagulation therapy or a mesenteric vascular occlusion. The patient made an uneventful recovery and follow-up study showed no abnormality of the small bowel.

Figure 1, *A* and *B*, Dr. Sanford Bluestein of Paterson, N.J., for Figure 2, *A* and *B*, Dr. Frank Glenn of Glens Falls for Figure 5, Dr. Donald Shiffman of Hackensack Hospital, Hackensack, N.J., for Figure 6, *A* and *B*, and Dr. A. Clemett, Yale University School of Medicine for Figure 10, *A* and *B*.

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UNUSUAL SMALL BOWEL DISEASES

METHODS AND OBSERVATIONS

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IN RECENT reviews the many etiologies associated with gastrointestinal malabsorptive syndromes are emphasized.[5,18,21,25,38] These can be classified on the basis of major defects in digestion and transport of fat (Table I, Volwiler,[38] modified). An equally useful classification divides malabsorptive syndromes into "primary" (group 3 of division V) and "secondary" forms. Steatorrhea is the most striking feature of malabsorptive syndromes regardless of etiology, if one excludes the rare instances of selective absorptive defect for a single aliment.

Mackie,[24] in 1933, and Snell and Camp,[35] in 1934, first called attention to the roentgenographic changes in steatorrhea. In subsequent reports, there has been unpredictable variation in the occurrence rate and in the severity and character of the roentgenographic changes in malabsorptive disorders.[18] Technique of examination appears to have been a major factor contributing to this inconsistency.

The present report concerns (a) case examples of 3 unusual diseases which feature malabsorption and roentgenographic alterations of the small bowel, and (b) observations regarding method of examination and interpretation of small bowel roentgenograms.

REPORT OF CASES

CASE I. An $8\frac{1}{2}$ year old white male was admitted to the Clinical Research Center of the University of Washington Hospital for study. Since birth, the patient has exhibited diarrhea, steatorrhea and retardation of growth and development. At $2\frac{1}{2}$ years of age, diagnoses of celiac syndrome and acanthocytosis were made. Muscular coordination had been impaired since age 4. At 6 years of age, small bowel biopsy specimens showed vacuolization of intestinal absorptive cells which were thought to contain excess lipid. Intermittent fecal incontinence and the inordinate ability to retain urine had been present for approximately 2 years prior to admission.

Physical examination revealed a somewhat dark-skinned white male with thin extremities and a moderately protuberant abdomen. Blood pressure, pulse rate, temperature and respiration were normal. The abdomen was not tender, and bowel sounds were normal. Evidence of posterior column and cerebellar dysfunction was found on neurologic assessment. The optic fundi were normal.

Results of extensive laboratory investigation included the demonstration of acanthocytosis, steatorrhea, low levels of plasma lipid, absence of plasma beta-lipoproteins and increased lipid content in small bowel mucosal biopsy specimens. Normal blood cells and plasma lipids were demonstrated in the parents.

Roentgenologic examination of the small bowel revealed thickening of mucosal folds and minimal flocculation of contrast material (Fig. 1, *A* and *B*).

Comment. Congenital absence of beta-lipoprotein results from an inborn error of metabolism as part of a disease which has acanthocytosis, neurologic ataxia, atypical retinitis pigmentosa, steatorrhea, retardation of growth and development, and extremely low levels of plasma lipids as its outstanding features.[40] Fewer than 10 cases have been reported.

Bassen and Kornzweig[4] in 1950 described the first case of a new syndrome. Their patient showed a spiny appearance of the red blood cells, retinopathy, and central nervous system disease. Singer *et al.*[34] in 1952 reported a case, and designated the morphologic aberration of the red blood cells "acanthrocytosis" (sic) (acanthus: thorn or sharp point). In 1960 Salt *et al.*[32] first reported the absence of plasma lipoprotein, which they considered the primary genetic defect. Family studies suggest that the disease is the manifestation of an autosomal gene in the homozygous state.

Intestinal malabsorption becomes mani-

TABLE I

A CLASSIFICATION OF MALABSORPTIVE DISORDERS

- I. Inadequate mixing of food with bile salts and lipase
    - 1. Pyloroplasty
    - 2. Gastroenterostomy
    - 3. Gastrectomy
- II. Inadequate emulsification of fat—lack of bile salts
    - 1. Obstructive jaundice
    - 2. Severe liver disease
- III. Inadequate lipolysis—lack of lipase
    - 1. Mucoviscidosis
    - 2. Chronic pancreatitis
    - 3. Neoplasm of pancreas or ampulla
    - 4. Pancreatic fistula
    - 5. Severe protein deficiency
- IV. Lymphangiectasia
    - 1. Inflammatory
    - 2. Neoplastic
    - 3. Idiopathic
- V. Defects of small intestinal absorption
    - 1. Malabsorption associated with blind loops, diverticula, stricture or fistula
    - 2. Inadequate absorbing surface
        - (a) inflammatory: regional enteritis, tuberculosis
        - (b) neoplastic
        - (c) amyloid infiltration
        - (d) progressive systemic sclerosis
        - (e) Whipple's disease
        - (f) resection
    - 3. Primary biochemical dysfunction of epithelial cells
        - (a) celiac sprue
        - (b) tropical sprue
        - (c) severe starvation
        - (d) transient dysfunction associated with intestinal infection
        - (e) a-beta-lipoproteinemia syndrome
    - 4. Drugs
    - 5. Vascular lesions
    - 6. Parasitic infestations

fest in infancy. The neurologic and ocular symptoms may begin from early childhood to early adolescence, and thereafter progress slowly and relentlessly.

The appearance of the intestinal mucosa is one of well-defined villi with vacuolated absorptive cells. These contain excessive amounts of lipid. Reports of the small bowel roentgenograms describe the nonspecific features observed in association with steatorrhea.[20,32]

The diagnosis of a beta-lipoproteinemia should be particularly suspected in patients with steatorrhea if serum cholesterol levels are less than $\frac{1}{5}$ to $\frac{1}{10}$ normal. Inspection of the blood in its fresh, undiluted state is necessary for demonstration of acanthocytes. The absence of beta-lipoprotein is confirmed by electrophoretic, immunochemical or ultracentrifugation techniques.[32] Available methods of therapy have been unsatisfactory.

The case above, manifesting absence of plasma beta-lipoprotein, acanthocytosis, neurologic disease, steatorrhea, retardation of growth and development, and low levels of plasma lipids, differs from those previously reported only in the absence of retinopathy.

CASE II. A 13 year old white male was admitted to the University of Washington Hospital because of diarrhea since birth and growth retardation. Birth weight was normal. During the first 6 months of life, he exhibited diarrhea several times daily. From 6 months of age until the time of admission, he produced 6 or 7 fatty stools each day. During recent years, the patient experienced several acute, well-documented episodes of hypocalcemia. Intermittent, painless abdominal distention was a recurrent feature of his illness. History of fever, anemia or melena could not be elicited. Family history was not contributory.

Physical examination revealed an alert, small white male in no distress. Blood pressure, pulse rate, temperature and respiration were normal. The abdomen was protuberant, tense and nontender. The liver was not enlarged. An ill-defined, freely movable mass of unspecified dimensions was palpable in the periumbilical area. Bowel sounds were normal. Genitalia were pre-pubertal and the neurologic examination was within normal limits. Peripheral edema was not observed.

Laboratory data included a normal hemogram, urinalysis and erythrocyte sedimentation rate. Numerous blood chemical determinations yielded normal findings, as did skin testing for tuberculosis and fungal diseases, sweat chloride test and duodenal content enzyme assay. Low levels of total serum protein, albumin and gamma globulin were observed by protein electrophoresis. Repeated stool examinations documented steatorrhea and the absence of pathogenic microorganisms. Peroral small bowel biopsy showed normal mucosa. Evaluation of gastrointestinal protein loss is planned for the future. Because of the ill-defined abdominal mass, laparotomy was performed and the following were described: absence of fluid in the peritoneal cavity, a diffuse mass-like thickening of the small bowel mesentery, a white pebbly appearance of the ileal mucosa and small yellow-white nodules near the mesenteric border. Multiple biopsies were performed. The histologic sections were considered diagnostic of intestinal lymphangiectasia as described by Waldmann *et al.*[39]

Roentgenograms of the small bowel showed mild dilatation and, in addition, a distinctive diffuse pattern of mucosal fold thickening which was different in character from that seen in celiac sprue (Fig. 2, *A* and *B*). The folds were not only thickened but appeared much more numerous than in a corresponding length of bowel in sprue. The skeleton was demineralized and minimal rachitic changes were observed.

Comment. Hypo-albuminemia is a common finding in a variety of diseases, including many intestinal disorders. In most of the latter, a mucosal lesion is demonstrable. In the absence of renal, hepatic, endocrine and obvious gastrointestinal disease, some instances of hypoproteinemia have been termed "idiopathic." It was in such a group of 12 patients studied by Waldmann *et al.*[39] that gross and histologic evidence of dilatation of intestinal and mesenteric lymphatics was found. The designation "intestinal lymphangiectasia" was suggested for this disorder which involved gastrointestinal protein loss. Waldmann *et al.* state that the diagnosis should be based on biposy or autopsy evidence. Although the term implies a characteristic histopathology, etiology and pathogenesis remain obscure. In no instance was there evidence of a neoplastic or inflammatory lesion to explain lymphatic dilatation. However, similar cases have been reported in which a nonspecific granulomatous lesion was found.[14] The oldest patient reported by Waldmann *et al.* was 28 years of age, and 5 patients among the 12 experienced onset of symptoms before the age of 10 years. Salient clinical features in this syndrome were edema at some time during the course of the disease, diarrhea, steatorrhea, hypo-albuminemia and hypogammaglobulinemia. Excessive

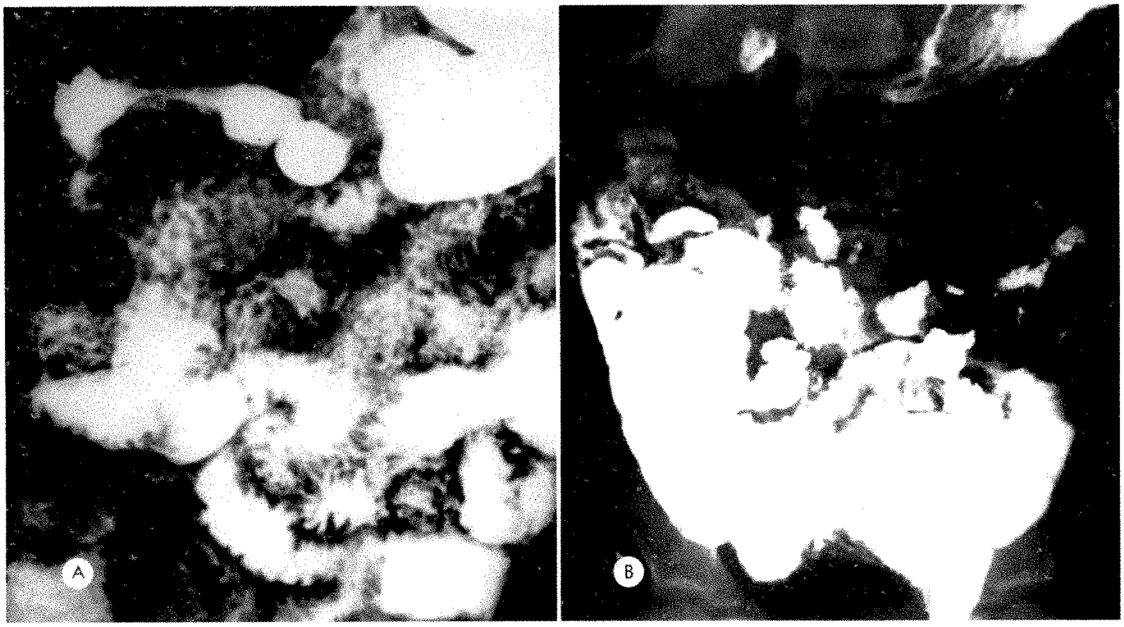

FIG. 1. Small bowel examination in a-beta-lipoproteinemia. (*A*) Barium fills the stomach, duodenum, jejunum and part of the ileum. Thickening of mucosal folds is especially prominent in the jejunum. (*B*) A roentgenogram 45 minutes later. Dilatation of several loops of ileum is shown on this as well as on *A*.

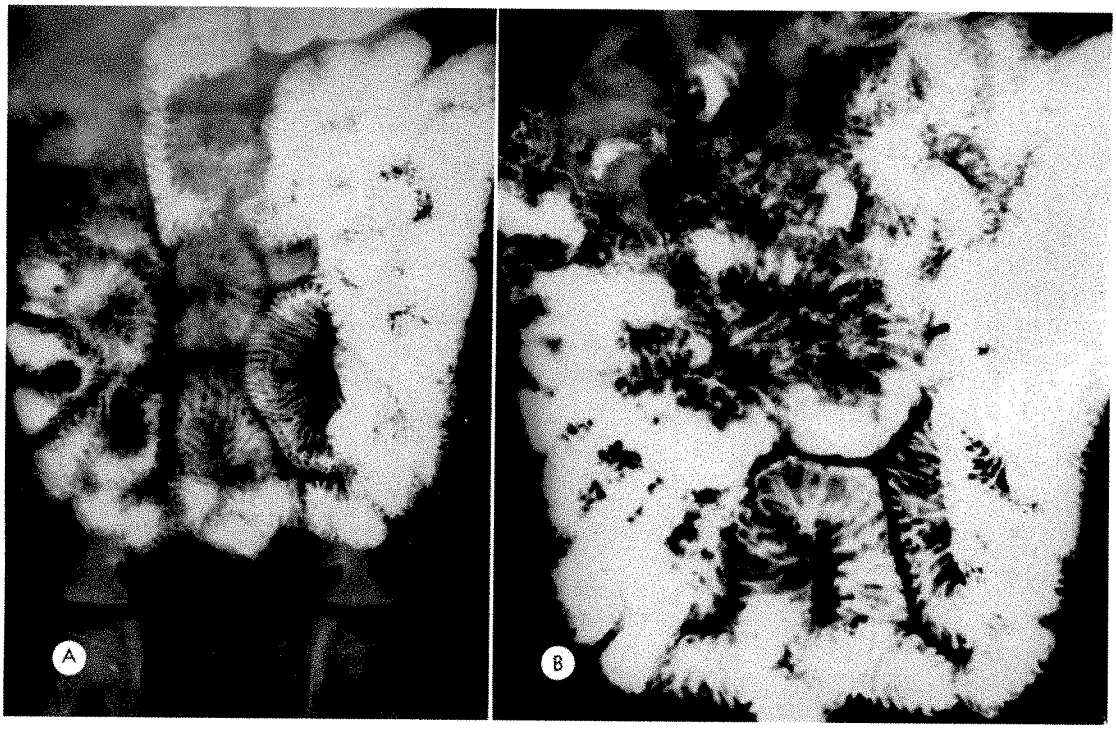

Fig. 2. Small bowel examination in lymphangiectasia. (*A*) Dilatation and thickening. Intestinal mucosal folds are prominent in (*A*) the jejunum and (*B*) the ileum.

loss of protein from the gastrointestinal tract was demonstrated in each case. Roentgenologic assessment of the small bowel revealed widening of the mucosal folds and flocculation in 9 of 12 patients; 3 were normal. Efforts at therapy including gluten-free diets, segmental small bowel resection, and therapeutic trials of corticosteroids did not result in favorable clinical response.

The case above is submitted as an example of intestinal lymphangiectasia. Dr. Waldmann has reviewed the clinical history and pathologic material in this case. He considers the histologic sections of the small bowel to present a picture typical for the disorder described by him and his coworkers.[40]

Case III. A 43 year old white male was admitted to the University of Washington Hospital because of a weight loss of sixty pounds and severe diarrhea during the 8 months prior to admission. He had been in good health except for an episode of "colitis" 14 years previously. Weight loss had occurred despite high caloric intake. Stools had been watery, foul, and often numbered 10 per day. The patient denied past or recent treatment with neomycin or other antibiotic medication.

Physical examination revealed an acutely ill, emaciated, agitated white male. Blood pressure was 100 systolic, 60 diastolic. Pulse, respiration and temperature were normal. The abdomen was moderately distended; neither the spleen nor the liver was enlarged; bowel sounds were normal. There was no peripheral edema, and neurologic deficit was not observed.

Laboratory assessment demonstrated macrocytic anemia, normal urinalysis and a slightly megaloblastic bone marrow. Relevant blood chemical values included low protein and low albumin. The serum carotene level was depressed and the prothrombin time was prolonged. The stool contained excessive fat; no blood was present in the stool. Vitamin B-12 absorption was normal. Serial peroral small bowel biopsy specimens have been interpreted as consistent with celiac sprue.[29,30]

Roentgenologic examination of the small bowel demonstrated marked dilatation and several large jejunal diverticula (Fig. 3, *A*, *B* and *C*).

Immediate clinical improvement followed institution of a gluten-free diet. However, corticosteroids and antibiotics were subsequently utilized to achieve maximal rate of improvement.

Comment. Jejunal diverticula are atonic mucosal sacs which herniate through the submucosa and muscularis. The incidence of jejunal and ileal diverticula demonstrated roentgenologically approximates 0.5 per cent.[28] Their detection as an incidental finding and the discrepancy between their reported incidence and the incidence of associated malabsorption have led to the concept that they are seldom of clinical significance.[22] This concept has recently been challenged by Cooke *et al.*[7] Stagnation of bowel contents with bacterial proliferation seems the most plausible explanation for the malabsorptive state which may be associated with jejunal diverticula.[27] That diverticula occur more frequently in older age groups suggests that they are acquired, although their pathogenesis remains obscure.

Steatorrhea was found in 9 of 27 patients reported by Cooke *et al.*[7] to have jejunal diverticulosis. Of these 9, 3 had small bowel biopsies, none of which showed the histo-

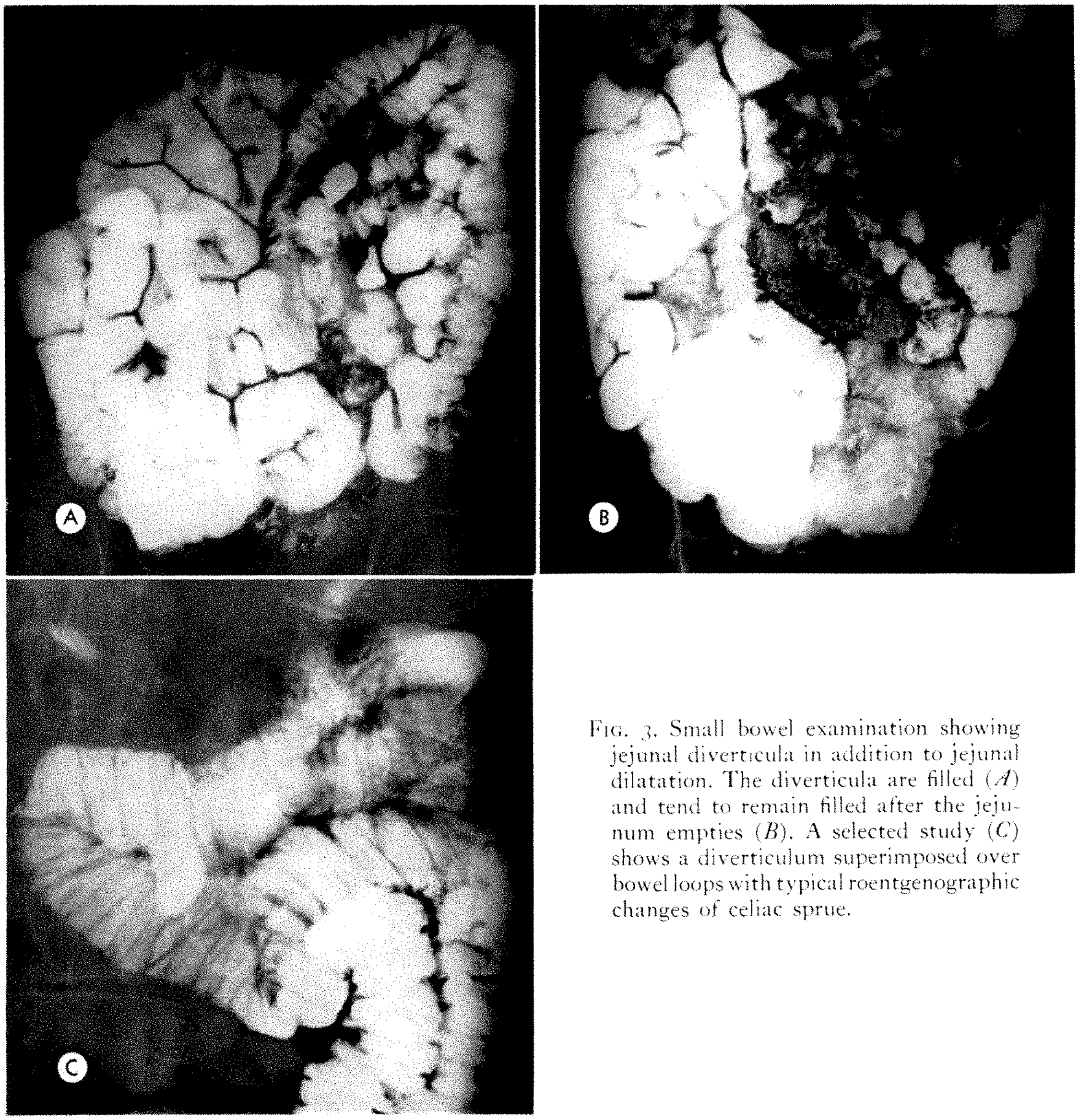

FIG. 3. Small bowel examination showing jejunal diverticula in addition to jejunal dilatation. The diverticula are filled (*A*) and tend to remain filled after the jejunum empties (*B*). A selected study (*C*) shows a diverticulum superimposed over bowel loops with typical roentgenographic changes of celiac sprue.

logic lesion of celiac sprue. It is surprising that only in one earlier case[33] has the histologic sprue lesion been associated with diverticulosis of the jejunum.

The case above is of unique interest because of the rarity of the associated conditions. In particular, it emphasizes the importance of roentgenologic examination despite the availability of an acceptable method of histologic diagnosis of sprue and other generalized small bowel mucosal lesions.

DISCUSSION

CONTRAST MATERIAL

In 1936, only 3 years after the initial descriptions of small bowel roentgenologic alterations associated with steatorrhea, Pendergrass *et al.*[26] reported differences in transit time and roentgenologic small bowel pattern as a function of several variables, including amount and consistency of contrast material. On this basis, these workers presented a plea for standardization of technique in examination of the stomach and small bowel. During the next 15 years, various terms were introduced to describe the appearances of barium sulfate when flocculated within the small bowel.[8,16] In 1943, Krause and Crilly[19] cited the many disease entities featuring the "deficiency pattern," and in 1945, Ruffin *et al.*[31] were the first to present evidence that the deficiency pattern could be demonstrated in apparently normal individuals. It was shown, in addition, that flocculation could occur in normal children,[37,41] and that it could be induced in normal subjects by the presence of mucus and fatty acids in the small bowel.[10] The pattern of flocculated barium sulfate in these instances was indistinguishable from that previously designated "deficiency pattern," "segmentation," or "disordered motor function."

The development of barium preparations partially stabilized against flocculation enabled British workers during the past decade to define more precisely the roentgenologic features associated with steatorrhea. Using such preparations, they observed what are now considered the most reliable anatomic correlates of steatorrhea: dilatation of the small bowel and thickening of transverse mucosal folds.[2,11]

More recently, Laws *et al.*[22] have reported their experience in relating roentgenologic to clinical histologic findings in 28 patients with celiac sprue. Occurrence or degree of flocculation correlated poorly with clinical status, amount of fecal fat excreted, and severity of villous atrophy. Conversely, jejunal dilatation to a caliber measuring greater than 30 mm. on the roentgenograms provided good correlation with clinical status and degree of steatorrhea. Thickened mucosal folds were seen in most of the patients with dilated jejunum, although no attempt was made to measure actual fold thickness. These observations are supported by personal experience with analysis of jejunal caliber and mucosal fold thickness in 16 cases of histologically documented celiac sprue.[9]

The majority of authors who have cited the volume of barium suspension utilized in roentgenologic examination of the small bowel indicate that volumes of 8 to 10 fluid ounces have been used most frequently.[10,13,15,17,19,26,31] Marshak[25] has stated his preference for volumes larger than those mentioned above. Recently, Caldwell and Floch[6] systematically studied 8 versus 16 ounces of barium, and reported faster transit and easier interpretation with the larger amount.

OBJECTIVES OF EXAMINATION

Malabsorptive syndromes can be detected roentgenologically in many instances. However, more sensitive and accurate methods of detection are available. The nonspecific findings of jejunal dilatation and thickening of mucosal folds will be encountered principally in steatorrhea associated with celiac sprue, tropical sprue, and more rarely, the infiltrative diseases of the small bowel, *e.g.*, Whipple's disease, scleroderma, lymphoma and amyloidosis. In the infiltrative diseases, additional roentgenologic abnormalities may be present. Jejunal dilatation and mucosal fold thickening can be expected less frequently in steatorrhea associated with hepatobiliary disease, exocrine pancreatic insufficiency and gastrectomy.

The aim of roentgenologic examination of the small bowel should be excellent anatomic delineation in detail comparable to that sought in the remainder of the gastrointestinal tract. Only this degree of precision will permit accurate assessment of jejunal caliber and mucosal fold thickness. It seems inconsistent that flocculation could be considered desirable, since it results in poor anatomic definition. Moreover, the flocculation phenomenon, incompletely understood and controlled with plain barium and water mixtures, correlates poorly with steatorrhea. Precise anatomic delineation is imperative in those instances where steatorrhea is caused by a localized or segmental condition such as regional enteritis, diverticulosis, strictures and internal fistulae.

The evidence cited above, as well as our personal experience, suggests that this can be best accomplished by a method of examination consisting of: (1) oral administration of a large volume (16–20 fluid ounces) of a flocculation-resistant barium suspension, (2) high kilovoltage roentgenograms of the abdomen made at frequent intervals, and (3) fluoroscopic examination and supplemental spot roentgenography performed at intervals indicated by review of the serial films of the abdomen.

Use of large volumes of contrast medium affords more rapid opacification of the entire small bowel. This results in several studies of any given segment of the small bowel as well as a significant decrease in time required for the examination. Superimposition of opacified adjacent loops is readily overcome by appropriate fluoroscopic manipulation and compression spot roentgenography.

With the method of examination described above, demonstration of the most reliable roentgenologic features of primary malabsorption syndromes, *i.e.*, dilatation and mucosal fold thickening, as well as the precise definition of pathologic anatomy, may be achieved optimally.

SUMMARY

Cases of 3 unusual diseases featuring intestinal malabsorption are presented together with small bowel roentgenograms: (1) a-beta lipoproteinemia with acanthocytosis; (2) lymphangiectasia of the small bowel; and (3) jejunal diverticulosis combined with celiac sprue. These are related to a general classification of malabsorption disorders. The particular clinical and roentgenologic features of these diseases are discussed.

The authors feel that the most satisfactory technique for roentgenologic examination of the small bowel is one where a large volume of flocculation resistant barium suspension is employed. This has the advantage of quick and complete roentgenography and permits optional anatomic definition. Flocculation is diagnostically useless and is to be avoided.

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EXPERIMENTAL INFARCTION OF SMALL BOWEL IN DOGS*

ROENTGENOGRAPHIC DEMONSTRATION WITH WATER-SOLUBLE IODINE CONTRAST MEDIUM

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PLAIN roentgenograms of small bowel infarction secondary to mesenteric vascular occlusion are usually inconclusive. They frequently show evidence of paralytic ileus, intraperitoneal free fluid, or complete absence of gas in the intestine.[3,4,8] Studies with barium enema may show complete collapse of the colon and a dilated small bowel in which air-fluid levels resemble mechanical obstruction. Frimann-Dahl,[1] Wang and Reeves,[7] and Samuel[5] have reported various findings with barium meal examination in mesenteric vascular thrombosis. Hibbard *et al.*[2] were unable to differentiate on plain roentgenograms between mesenteric vascular occlusion and mechanical small bowel obstruction in dogs.

In order to study the effects of small bowel infarction, vessels supplying blood to various parts of the small intestine were ligated in dogs and oral, water-soluble contrast medium was employed for the roentgen examination. Such a medium is used in patients with acute abdominal conditions in preference to barium sulfate, because of faster transit time. It is also safer if perforations are present.

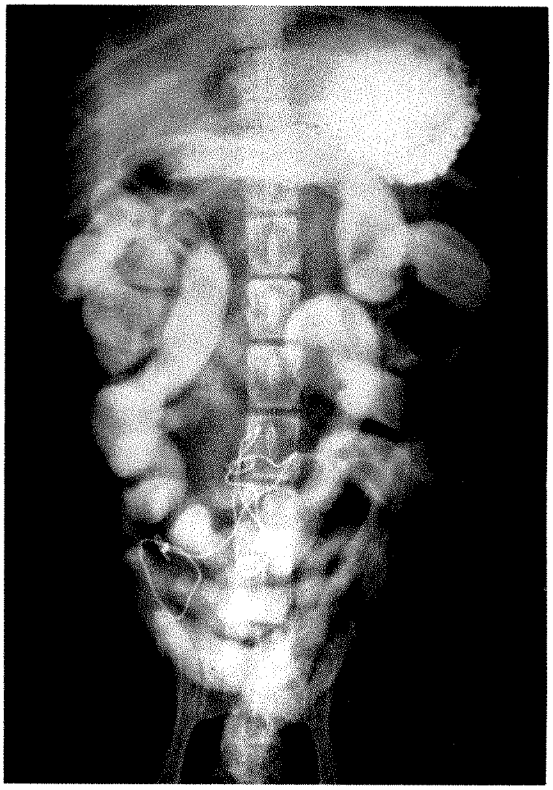

FIG. 1. Dog 1. Normal small bowel appearance on eighth day after ligation of arteries supplying 17 cm. ileum, 20 cm. from ileocecal valve.

MATERIAL AND METHOD

The ileal arteries to a small segment of ileum were ligated in 2 dogs without resultant infarction of the small bowel (Fig. 1). In an additional 11 dogs, in which the blood supply of various segments of the small bowel was occluded, infarction was induced (Table 1) (Fig. 2 through 6).

Preoperative roentgen studies were made of the transit time required for 100 cc. of contrast medium to pass from the stomach to the cecum. The contrast medium, prepared in a 30 per cent solution from powdered hypaque (diatrizoate sodium), was administered through a Levin tube into the stomach. Serial roentgenograms were made until the head of the column of the contrast medium reached the cecum.

Each dog was anesthetized with an intravenous solution of 1.2 per cent sodium pentothal. Aqueous procaine penicillin, 600,000 units, was given intramuscularly

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Table I

ROENTGEN STUDIES OF EXPERIMENTAL INFARCTION OF SMALL BOWEL IN DOGS

	Occluded Vessel and Location	Roentgen Studies with Hypaque	Small Bowel Segment at Time of Resection
Group A—Arteries, Lower Ileum			
Dog 1	17 cm. ileum, 20 cm. from cecum	Normal 8 days after ligation	Grossly and histologically normal on 21st postoperative day
Dog 2	15 cm. lower ileum	Normal on 4th and 7th postoperative day	Normal on 8th postoperative day
Group B—Superior Mesenteric Artery			
Dog 3	Below origin of right colic artery	Delayed transport time; edematous, infarcted bowel with accordion-like contractions increasing for 72 hr.	Third postoperative day, 80 cm infarcted bowel
Dog 4	Above right colic artery	Immediate accordion-like contraction rings of small bowel shown by prior small bowel hypaque study	Died 48 hr. after ligation from perforated lower ileum
Group C—Arteries and Veins, Lower Ileum			
Dog 5	10 cm. of small bowel, 15 cm. proximal to cecum	Segment proximal to infarction edematous; partially contracted small bowel with contraction rings 9 hr. after infarction	10 cm. slightly discolored ileum 26 hr. after ligation; slight smoothness of infarcted segment; contraction rings of pre-infarcted and post-infarcted segment
Dog 6	20 cm. of small bowel, 16 cm. proximal to cecum	Sixteen hr. after ligation, edematous contracted proximal segment with accordion-like rings	17 cm. infarcted bowel 25 hr. after ligation; findings similar to Dog 5
Dog 7	20 cm. of ileum, 15 cm. proximal to cecum	Seven hr. after hypaque and 11 hr. after ligation, infarcted segment with typical changes	Minimal increased tone and discoloration of 10 cm. segment of ileum 11 hr. after infarction
Dog 8	12 cm. of ileum, 20 cm. proximal to cecum	Nine hr. after hypaque and 12 hr. after infarction, accordion-like contracted proximal segment and slightly dilated infarcted segment	Ileum, necrotic 15 cm. 30 hr. after ligation
Dog 9	15 cm. of ileum, 30 cm. proximal to cecum	Eight hr. after hypaque and 9 hr. after ligation, early changes with slight circular contractions of proximal segment	A 12 cm. infarcted segment 30 hr. after ligation; proximal segment shows rippled contraction rings
Dog 10	18 cm. of ileum, 20 cm. proximal to cecum	Seven hr. after hypaque and 9 hr. after ligation, typical contracted proximal segment in lower ileum	A 15 cm. infarcted, edematous bowel segment with proximal segment contraction rings and mucosal ulcerations of infarcted segment

(*Continued on next page*)

TABLE I (*Continued*)

	Occluded Vessel and Location	Roentgen Studies with Hypaque	Small Bowel Segment at Time of Resection
GROUP D—Arteries and Veins, Midportion of Small Intestine			
Dog 11	15 cm. of upper ileum, 80 cm. proximal to cecum	Vomited hypaque 3 times; contrast medium failed to leave stomach; bladder visualized	Died 16 hr. after ligation; perforated small bowel at autopsy
Dog 12	15 cm. of ileum, 100 cm. proximal to cecum	Vomited hypaque twice; contrast medium failed to pass beyond stomach	Died 14 hr. after ligation; perforated small bowel at autopsy
Dog 13	20 cm. of small bowel, 75 cm. proximal to cecum	Vomited hypaque 3 times over a period of 7 hr. without contrast medium leaving the stomach; bladder visualized	16 cm. black necrotic bowel 6 hr. after ligation

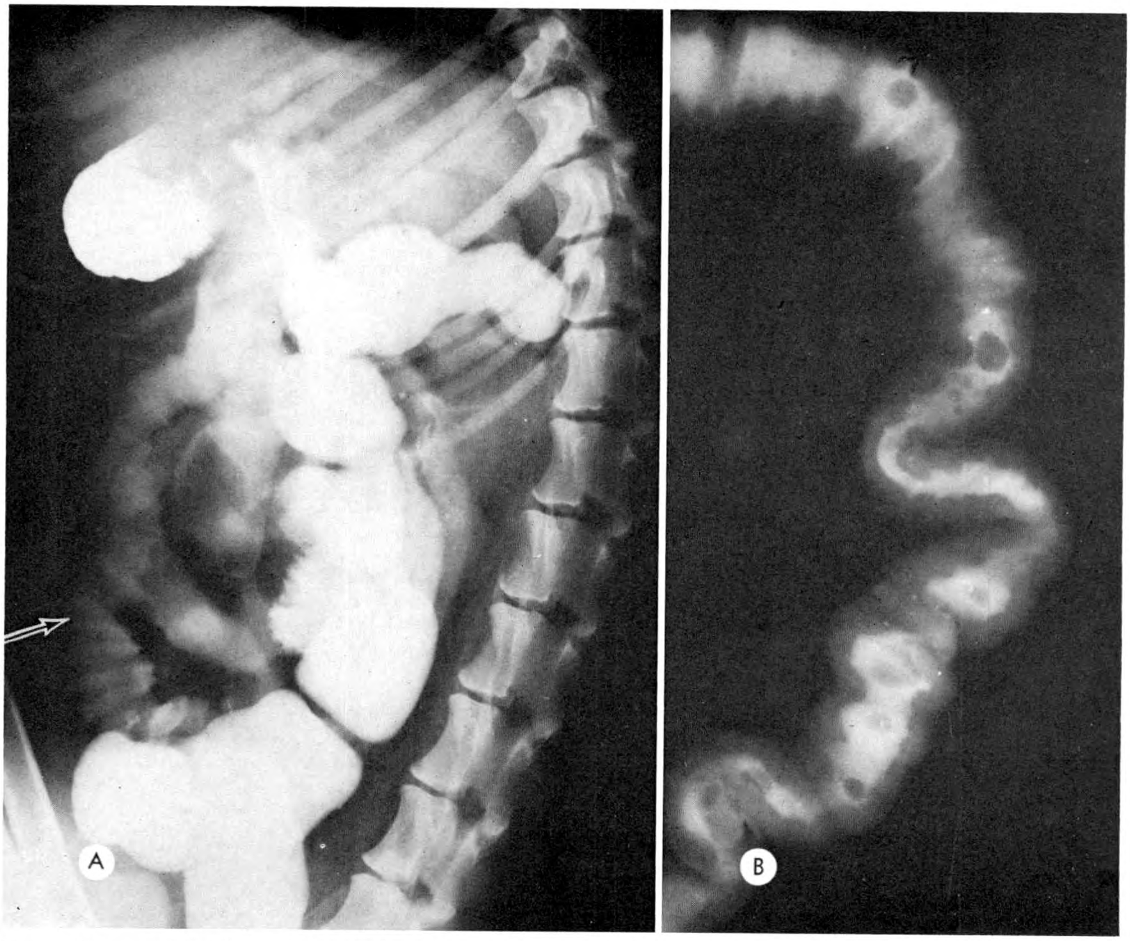

FIG. 2. Dog 3. (*A*) Hypaque small bowel study 48 hours after ligation of superior mesenteric artery. (*B*) Spot roentgenogram of infarcted bowel showing edematous bowel with accordion-like contraction rings.

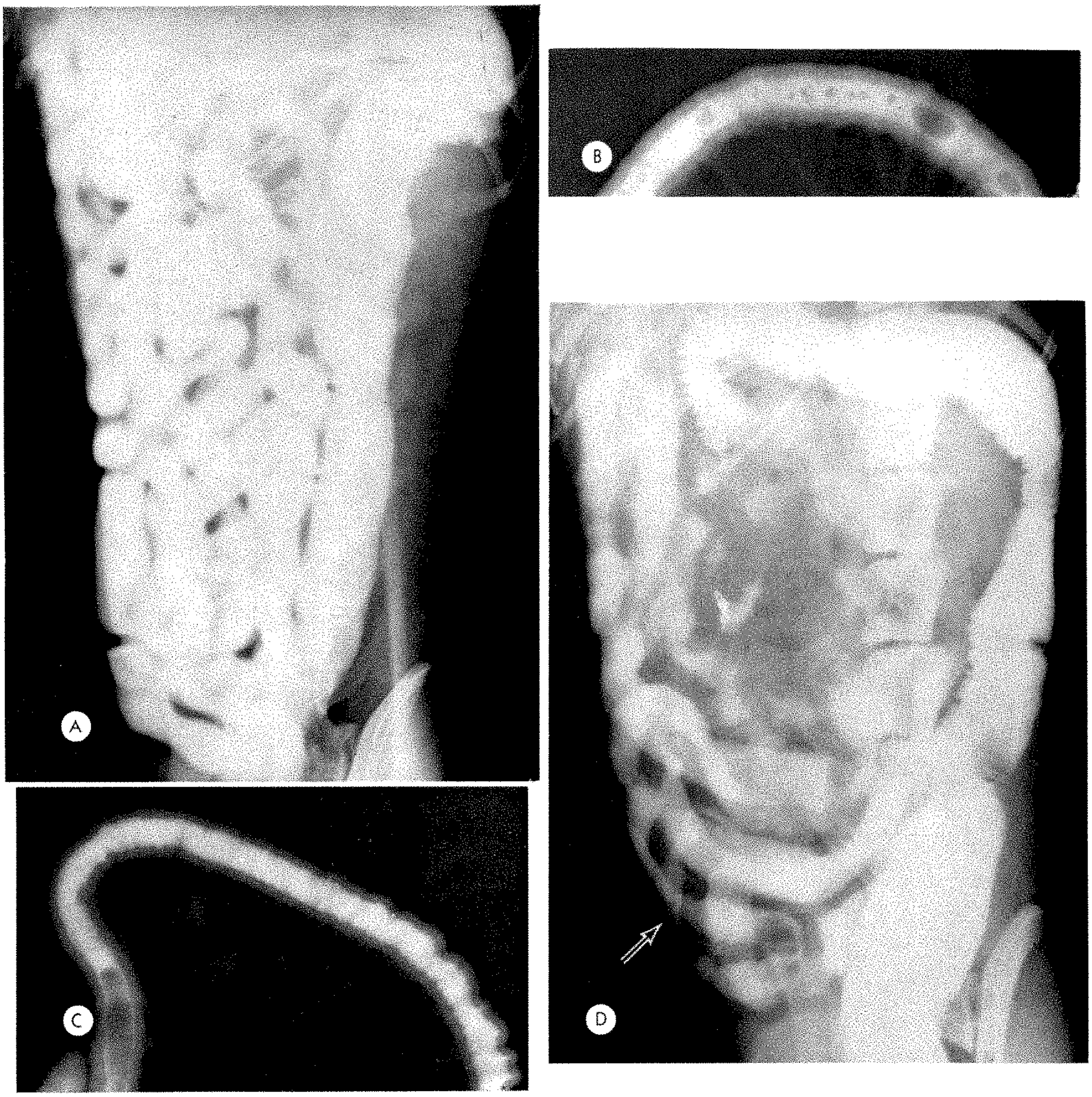

Fig. 3. Dog 4. (*A*) Normal preoperative small bowel study. (*B*) Spot roentgenogram of lower ileum immediately before ligation of the superior mesenteric artery. (*C*) Spot roentgenogram of same lower ileal region 10 minutes after arterial ligation. (*D*) Abdominal roentgenogram 25 minutes after arterial ligation showing contraction rings in small bowel.

daily during the experimental procedure, starting on the day that the blood supply to the small bowel was occluded.

On the first postoperative day, the hypaque solution was injected by tube into the stomach and serial roentgenograms were obtained. These were exposed as long as the contrast material remained in the stomach and until it was vomited or passed into the colon. If the animal survived the immediate postoperative period and roentgen study, a laparotomy was performed. The infarcted segment was roentgenographed and then resected. A roentgenographic transit-time study with hypaque was then done and compared with the studies done before the infarct was produced and with the infarcted segment in site.

Thirteen dogs were divided into 4 groups,

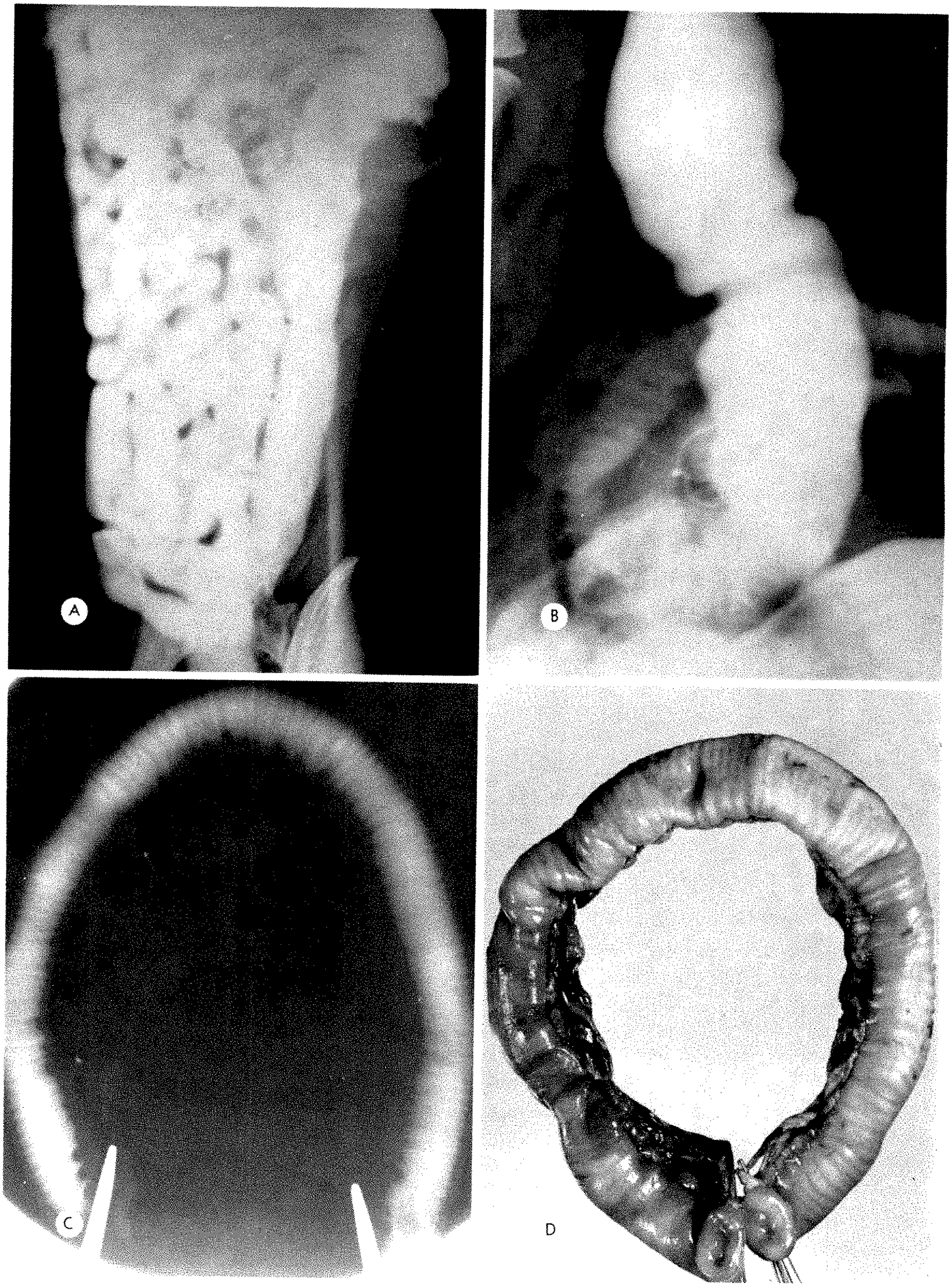

FIG. 4. Dog 5. (*A*) Normal preoperative hypaque small bowel study. (*B*) Spot roentgenogram 9 hours after infarction of 10 cm. segment of distal ileum. (*C*) Resected infarcted segment between opaque markers with proximal and distal segment contraction rings. (*D*) Photograph of resected segment.

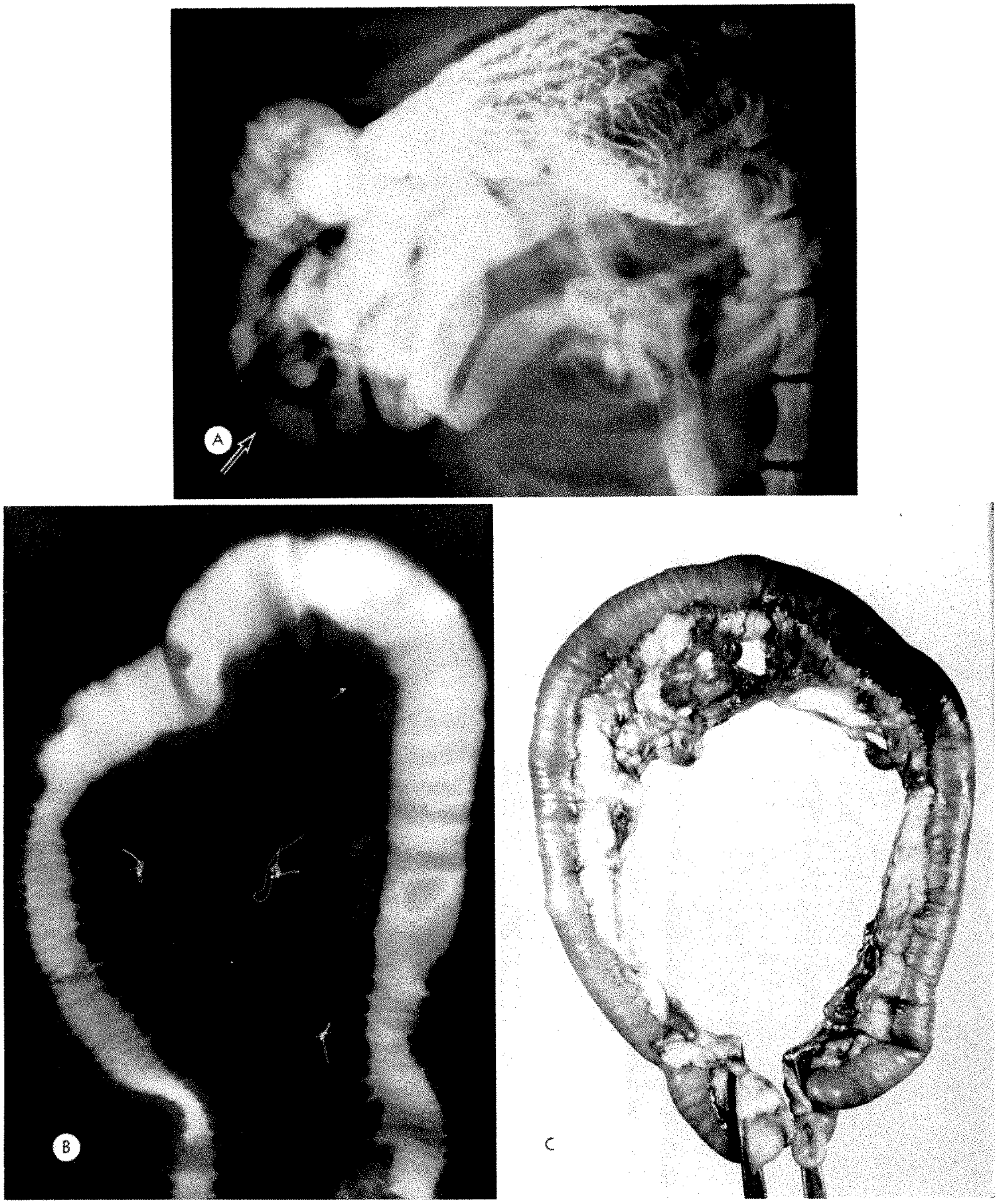

Fig. 5. Dog 7. (*A*) Nine hours after hypaque and 12 hours after infarction of 12 cm. of lower small bowel. (*B*) Resected segment. (*C*) Photograph showing proximal and infarcted areas.

A, B, C and D, according to the region of infarction and the results were evaluated (Table 1).

Group A. *Arterial ligation without infarction.* In Dogs 1 and 2, the simple arterial ligation of one of the ileal branches was low enough to permit collateral circulation and prevent infarction. No abnormalities of the bowel were demonstrated by hypaque study or at surgical exploration (Fig. 1). On

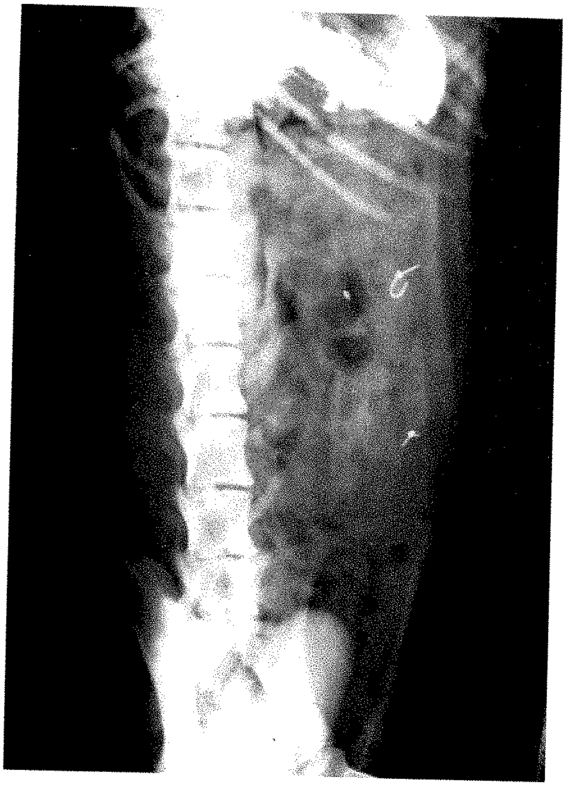

Fig. 6. Dog 11. Seven hours after ligation of 50 cm. of upper ileum. Vomited each time after receiving 100 cc. hypaque, which did not pass beyond stomach, although was retained long enough for absorption and bladder visualization.

histologic examination, the resected segments were normal.

Group B. *Ligation of superior mesenteric artery with infarction.* The superior mesenteric artery was ligated below the origin of the right colic artery in Dog 3 (Fig. 2, *A* and *B*). The bowel discolored gradually and massive infarction was demonstrated roentgenographically within 6 hours after ligation of the vessel. Edematous bowel with irregularly contracted areas and retention and delay in the transport of the contrast medium through the small bowel manifested this infarction. Similar, but progressive, changes were seen in the small bowel study with hypaque 48 hours after ligation (Fig. 2*A*).

Dog 4 (Fig. 3, *A–D*) had been given 100 cc. of hypaque solution via the Levin tube 90 minutes before ligation of the artery. After the contrast medium had passed into the lower ileum, the abdomen was opened and the superior mesenteric artery was ligated above the right colic artery. The effects of arterial obstruction were produced immediately and were easily shown by contrast medium already present in the bowel from the earlier oral administration.

Group C. *Ligation of arteries and veins of small segment of distal ileum.* An infarction was produced in a small segment (10 to 20 cm.) of the lower ileum in Dog 10. The main ileal branches of the ileocolic artery and of the collateral arteries at the mesenteric border of the bowel were ligated. An effect similar to arterial ligation was produced but hemorrhagic infarction occurred earlier.

In each dog the characteristic rippled or accordion-like circular contractions of the 10 to 30 cm. long afferent loop were visible roentgenographically, just proximal to the infarction. The area of infarction was usually seen.

Group D. *Ligation of arteries and veins of mid small intestine.* In Dog 11, an infarction was produced in a 15 cm. segment 80 cm. from the ileocecal valve. An immediate hemorrhagic infarction in a 15 cm. segment of the upper ileum was produced by this technique in Dog 12. A 20 cm. segment of the small bowel 75 cm. from the ileocecal valve was infarcted in Dog 13.

Emptying of the stomach into the small bowel was markedly inhibited in each dog of this group. Even after administration of the hypaque solution via the Levin tube, repeated vomiting occurred. Contrast medium, however, was retained long enough so that the bladder was visualized (Fig. 6). Roentgenograms of the infarcted segments which contained hypaque solution showed them to be irregularly dilated with evidence of mucosal ulcerations and bowel perforations. Typical accordion-like contractions were seen in the bowel wall of the segment proximal to the infarction.

The preoperative transport time of the hypaque solution from stomach to cecum was 90 to 180 minutes, with an average of 120 minutes. In Dogs 1 through 10, the transport time to the affected loop and through the small bowel was prolonged. The time required for the contrast medium to reach the cecum varied from 4 to 9 hours.

CONCLUSIONS AND SUMMARY

Ligation of the superior mesenteric artery or of the veins and arteries of the ileum at various points produced infarction of the small bowel in dogs. Roentgenographic examinations of these infarctions with oral, water-soluble contrast medium revealed the following characteristics: The transit time of the hypaque solution from the stomach to the cecum was delayed. Characteristic accordion-like contractions encircled the segment proximal to the infarction or the initially infarcted region. As infarction progressed, the infarcted segment became irregularly dilated and smooth. When the infarction was in a large segment or proximal to the distal ileum, the contrast medium was delayed in leaving the stomach. A similar delay or marked retention of the contrast medium in the stomach has been seen in closed-loop obstruction with strangulation of the blood supply of the small intestine.[6] These delays were related to the amount and point of infarction of the small bowel.

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THE MEANDERING MESENTERIC ARTERY OF THE COLON*

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IT IS generally accepted that the angiographic demonstration of collateral vessels always indicates vascular occlusion. Further, even though the actual site of the occlusion may not be visible, one can generally infer its location and extent from the pattern and direction of the flow in the collaterals. The "meandering mesenteric artery" of the colon represents such a collateral. We have encountered this large tortuous vessel only in association with occlusive abdominal arterial disease. Yet, its presence and significance are often not appreciated.

As with all collaterals, this artery is merely an enlargement of a normally existing vessel. It has been called by a variety of terms, including marginal artery, mesomesenteric artery, middle colic-left colic collateral, artery of Drummond, arc of Riolan, and arc of Treves. Unfortunately, as is so often true with eponyms in medicine, some authors have applied these names to other mesenteric vessels, and so the terminology has become confused. To add to the problem, there are actually 2 and sometimes 3 anastomotic arteries of the colon in the normal individual.

The purposes of the present communication are to clarify the terminology relating to the anastomotic arteries of the colon, to describe the roentgenologic features of the meandering mesenteric artery, and to emphasize its importance in the diagnosis of occlusive arterial disease.

NORMAL ANATOMY

The celiac artery arises from the aorta about 1.5 cm. above the superior mesenteric artery. Its gastric branches anastomose freely with those of the phrenic and esophageal arteries above, while its major linkage with the superior mesenteric artery is through its superior pancreaticoduodenal division. Frequently one finds a direct retropancreatic anastomosis from the celiac to the middle colic artery which has been called the anastomotic artery of Bühler.[9]

The superior mesenteric artery (Fig. 1) generally arises about 1.5 cm. above the left renal artery, in the vicinity of the first lumbar vertebra, and crosses the third portion of the duodenum. It gives off, more or less in sequence, the middle colic, inferior pancreaticoduodenal, right colic, ileocolic, and multiple small intestinal arteries. The right colic artery is not a constant vessel, sometimes arising from the ileocolic or middle colic artery. The middle colic artery terminates in right and left branches, 2 to 4 in number, which supply the transverse colon. It occasionally arises from the celiac rather than the superior mesenteric artery. Rarely it is completely absent, in which case the transverse colon is supplied by branches of the right and left colic arteries. In about two-thirds of normal individuals, there is a direct continuation of the left branch of the middle colic into the left colic artery. This continuous vessel, extending from the main trunk of the middle colic artery to that of the inferior mesenteric artery, we have called *the central anastomotic artery of the colon* (Fig. 1, no. 5).

In about 10 per cent of individuals, an accessory artery arises from the main branch of the superior mesenteric artery and extends directly to the middle colic or left colic artery, *i.e.*, to the central anastomotic artery.[16,21] We have called this vessel *the accessory anastomotic artery of the colon* (Fig. 2, *A–D*, no. 5a and 7a).

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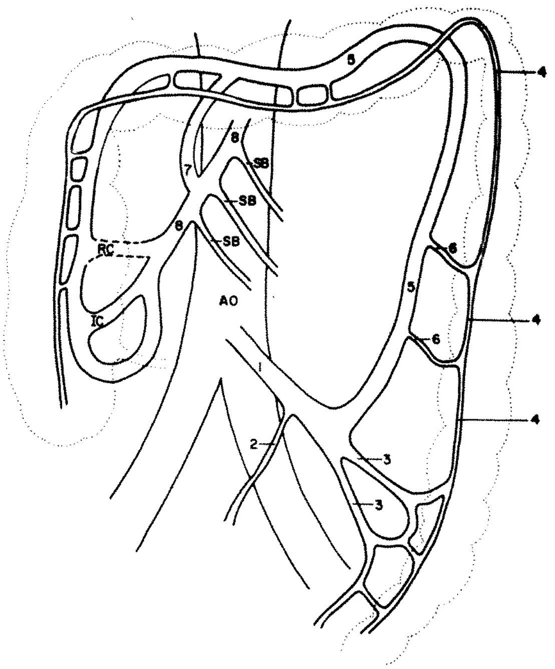

Fig. 1. Diagram of the normal anatomy of the mesenteric arterial circuit: (1) inferior mesenteric; (2) superior rectal; (3) sigmoid branches; (4) marginal artery of colon; (5) central anastomotic artery; (6) primary arcading branches; (7) middle colic; (8) superior mesenteric; (RC) right colic; (IC) ileocolic; (SB) small bowel branches; (AO) aorta. The inferior pancreaticoduodenal arcades and the celiac axis have been omitted for clarity. The marginal artery (4) is always closest to the colon. (The same numerical system is used in all illustrations.)

The inferior mesenteric artery, considerably smaller than the superior, arises from the left ventral surface of the aorta at about the level of the third lumbar vertebra. It gives origin to an ascending division—the left colic—and then continues downward, providing 1 to 3 sigmoid branches, and terminating in the superior rectal artery. The latter anastomoses with the middle rectal artery, a branch of the hypogastric artery. The left colic artery supplies the colon from approximately the region of the splenic flexure to the sigmoid. As stated above, it is usually continuous with the left branch of the middle colic, forming the central anastomotic artery.

Multiple transverse branches from the central anastomotic artery, as well as from the right colic and sigmoid arteries, supply an artery which parallels the entire colon near its mesenteric margin. This peripheral anastomotic artery is *the marginal artery* (Fig. 1, no. 4). It lies farthest from the colon wall at the points of bifurcation of the supplying arteries, and is usually situated closer to the colon on the left side than on the right.[18] The marginal artery is not always a continuous vessel. The segment in the region of the transverse and descending colon is rather constant, but continuity is said to be lacking in the region of the ascending colon in about 5 per cent of individuals, in the region of the sigmoid in 20 per cent, and at the rectosigmoid much more often.[18] Secondary arcades extend at irregular intervals from the marginal artery and give off the vasa recti, which terminate in the colon wall. Where the marginal artery is incomplete, the arcades arise from the central anastomotic artery. At times, the vasa recti arise directly from the parent artery without intervening arcades. The vasa have limited intramural connections and thus function as end-arteries.[13,18]

The blood supply of the colon, then, is derived from the ileocolic, the right, middle, and left colic, the sigmoid, and the rectal arteries. There are 2 relatively constant and 1 inconstant anastomotic arteries of the colon: the central, the marginal, and the accessory.

REVIEW OF LITERATURE

The normal interconnections between the celiac, superior mesenteric, inferior mesenteric, and hypogastric arteries have been studied by many anatomists and surgeons. Jean Riolan, a 17th century anatomist, was probably the first to point out the communications between the superior and inferior mesenteric arteries. He described the arterial arcade in the transverse mesocolon which has since been known by his name. His contribution was particularly intriguing because he was famous for his opposition to Harvey's theory of the circulation of the blood.[18] Treves, an Italian surgeon, described the intermesenteric anastomoses in

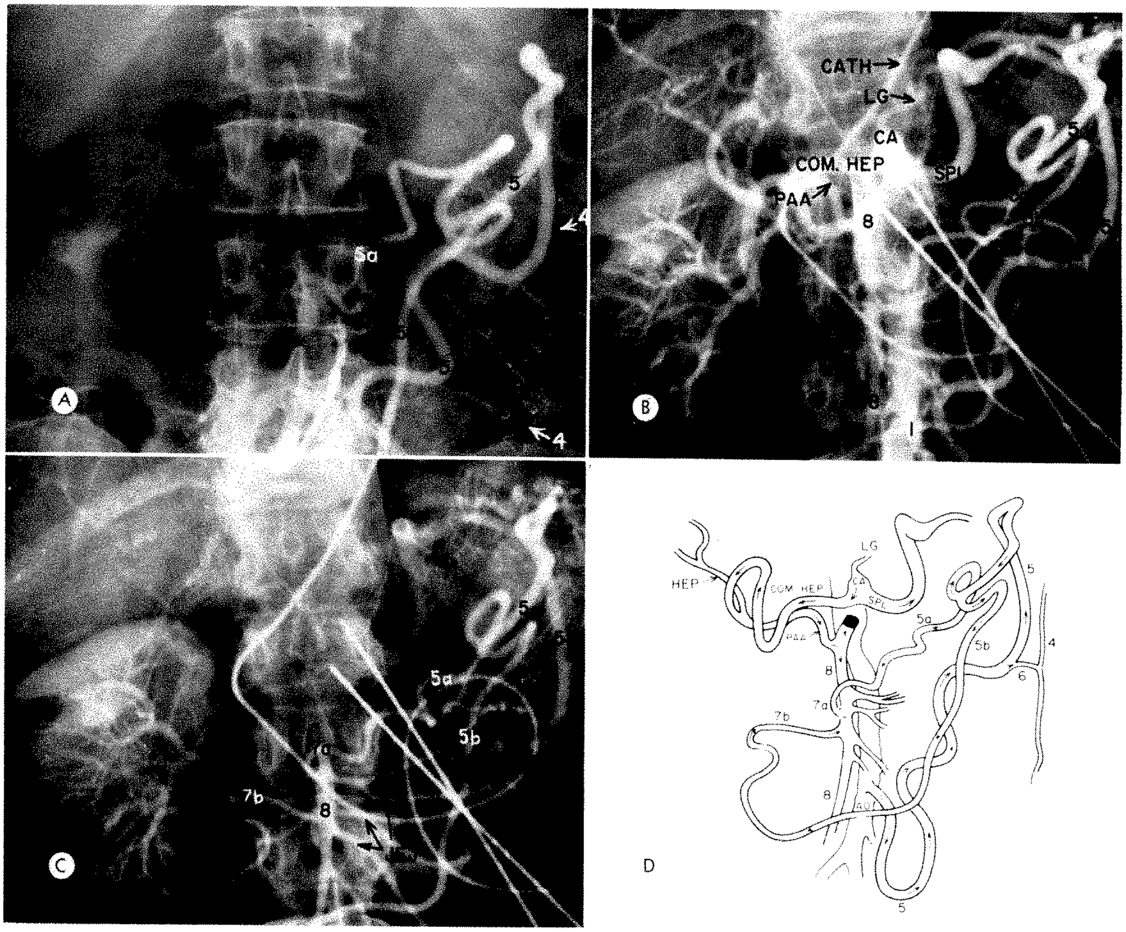

Fig. 2. Occlusion of the origin of the superior mesenteric artery. (*A*) Injection *via* catheter in distal aorta. The large meandering artery represents the central anastomotic artery of the colon (5). It gives off arcades (6) which form a normal-sized incomplete marginal artery (4). (*B* and *C*) Translumbar injection in the same patient, showing collateral flow from the celiac to the superior mesenteric artery (8) distal to the occlusion. An accessory anastomotic artery (5a–7a) is seen draining into the superior mesenteric from the central anastomotic artery near the splenic flexure. The middle colic (5b–7b) lies somewhat caudally because of low position of the transverse colon. (*D*) Composite drawing of *A*, *B* and *C*. Additional symbols: (CA) celiac artery; (Com Hep) common hepatic artery; (SPL) splenic artery; (LG) left gastric artery; (PAA) pancreaticoduodenal arcade; (JEJ) jejunal arteries. (Courtesy, Department of Radiology, Cincinnati Jewish Hospital.)

1898, and the arterial arborization in the right half of the mesocolon bears his name.[5] Sudeck, in 1907, ligated the superior rectal (superior hemorrhoidal) artery at its origin from the inferior mesenteric in cadavers. He then injected the main trunk of the inferior mesenteric artery with red lead and achieved retrograde filling of the superior rectal and its branches via the left colic and sigmoid arteries.[1] Cadaver ligation-injection techniques of this type were subsequently utilized by many others to study the mesenteric circulation.

The name most widely linked with the subject of mesenteric arterial circulation is that of Hamilton Drummond,[1] a British surgeon. In 1913, he described a series of roentgen angiographic studies in cadavers. Among other experiments, he occluded the proximal ends of the right, the middle, and the left colic and sigmoid arteries and injected the ileocolic artery with contrast medium. He demonstrated that all these vessels were connected. Examination of his 2 illustrations reveals both a central anastomotic artery of the colon and an incomplete marginal artery. Drummond called this entire anastomotic complex the marginal artery, and thus introduced some of the confusion in terminology. He also described the anastomoses between the superior and the middle rectal arteries. Although he credited others with prior demonstration of the marginal artery, many subsequent authors have termed one or both of the anastomotic arteries the *artery of Drummond.*

In 1933, Steward and Rankin[18] reported their extensive experiments with cadaver injections of the mesenteric circulation. They confirmed many of the earlier findings and, in addition, noted that in 63 per cent of individuals the left branch of the middle colic artery *directly* joined the left colic. They considered this central anastomotic artery a "large and more distally* situated marginal artery."

Williams and Klop[21] described another central artery in the root of the mesentery which joined the main trunk of the superior mesenteric to the middle or left colic artery. This vessel was found in 11 per cent of normal individuals. It is probably the same as the accessory middle colic artery described by Steward and Rankin,[18] and others, and is identical with our accessory anastomotic artery.

Recently, more and more has been written concerning the alterations that occur in these anastomotic vessels consequent to obstruction of a major mesenteric artery. Reiner *et al.*[14,15] performed postmortem injection studies in patients with occlusive arteriosclerosis of the celiac, mesenteric, or aortic circulation. They frequently encountered enlargement of one or more of the 3 anastomotic colic arteries. These colic vessels could be demonstrated in their entirety by injections into any of the main arteries which were still patent (celiac, superior mesenteric, or inferior mesenteric).

There are many reports of *in vivo* angiographic demonstrations of obstructing arterial lesions in the abdomen in which attention has been called to a large meandering artery in the mesentery which carries collateral flow to the colon.[2,3,5,6,17,19,20,22] Muller and Figley[10] noted that in aortic occlusion below the superior mesenteric artery, blood sometimes flowed from the superior mesenteric artery through a tortuous anastomotic artery into the inferior mesenteric and re-entered the aorta distally. The full importance of this meandering artery, however, has not been adequately emphasized in the literature.

ROENTGEN FINDINGS

In abdominal aortography performed in the normal individual by either percutaneous or catheter technique, the celiac and superior mesenteric arteries and their major branches are always demonstrable. At times, however, the inferior mesenteric artery, for no apparent reason, fails to fill. The smaller subdivisions of the mesenteric arteries, of course, are seldom as well demonstrated by aortic injection as by selective arteriography.

With studies of excellent quality, portions of the anastomotic arteries of the colon are often visualized. Although Pratt[12] states that the marginal artery is never seen in the normal individual, we have identified in nonoccluded cases short segments of the vessel and, on occasion, have observed more than half of its extent. The second-order divisions of the arteries to the colon (the left colic and the right and left branches of the middle colic) are often normally depicted. We have sometimes recognized the central anastomotic artery (Fig. 7) but have not identified the accessory artery in the normal state. Nor have we seen interconnections between the sigmoid and rectal vessels or the superior-middle rectal communications. However, the arcades connecting the superior and inferior pancreaticoduodenal arteries are not infrequently demonstrated.

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* Obviously they meant more distant from the colon.

In occlusive disease of the mesenteric arteries or aorta, the meandering mesenteric artery of the colon is usually present. It stands out as a bold, thick, wide vessel of uniform caliber pursuing a tortuous course around the abdomen and joining the superior and inferior mesenteric circulations (Fig. 2, *A–D*). Its caliber approaches and sometimes equals that of the parent artery. Although its course through the abdomen appears to be aimless, the vessel more or less parallels the mesenteric border of the colon, some distance away from its wall. It often dips quite low in the abdomen and may even disappear on the roentgenogram into the pelvis, only to reappear in the right lower quadrant. In such cases, it is merely following the course of a low lying transverse colon. We have not encountered a single instance in which a meandering artery extended down the sigmoid toward the rectum, nor have we seen a distinct example of continuity of the vessel along the cecum and ascending colon.

In all our cases in which the meandering artery was visible, it appeared to represent the central anastomotic channel rather than the marginal artery. The evidence for this is as follows: (1) the artery appears to arise and end relatively close to the main trunks of the superior and inferior mesenteric arteries; (2) it seems to be continuous with the middle colic and the inferior mesenteric (Fig. 2, *A–D;* 3, *A–D;* and 6, *A–C*), and there is no separate middle or left colic artery. The descending division of the inferior mesenteric artery does not appear to participate in the enlargement; (3) the meandering artery lies a greater distance from the colon than the marginal artery, and the arcades emanating from it are relatively long (Fig. 2 and 3); (4) in one instance we were able to demonstrate, in addition to the meandering artery, a second small, straight, continuous artery fed laterally from the meandering artery and ascending along the margin of the left colon to at least the distal transverse colon (Fig. 3, *A–D*). Although this represents the only example of extensive filling of the marginal artery in our abnormal cases, short segments of this vessel along the descending colon were commonly seen (Fig. 2, *A–D*). Invariably its caliber was small and it never appeared to represent a significant collateral pathway.

In 2 cases, discussed later, we have seen a bifurcation of the proximal end of the meandering artery. We believe one of these branches represented the accessory anastomotic artery (Fig. 2, *A–D*). At times the vasa recti have been visible and vascular "staining" of the colon wall has been noted.

The anastomotic arteries of the colon are, of course, not the only available channels for shunting blood to the colon. The pattern of collateral flow in the abdomen varies with the site of the obstruction. A description of some of these patterns follows.

OCCLUSION OF THE CELIAC ARTERY

We have had no personal experience with this condition although it has been described pathologically. While the phrenic, esophageal, and other thoracic arteries provide some of the collateral flow, most of it probably comes from the superior mesenteric artery through the pancreaticoduodenal arcades. Reports of isolated celiac occlusion are quite rare.[14,15] Reiner *et al.*[15] reported a case of combined celiac and superior mesenteric obstruction in which autopsy injection studies were performed. These indicated that the inferior mesenteric artery, via the anastomotic arteries of the colon, had maintained the circulation to both the celiac and superior mesenteric distribution so well during life that infarction was prevented. Rob and Owen,[16] during an operation for abdominal aneurysm, noted absence of blood flow in the main channels of the celiac and superior mesenteric arteries. These vessels were ligated and the aneurysm was removed. A postoperative aortogram showed a very large meandering artery arising from the inferior mesenteric and supplying the superior mesenteric and celiac branches. Mesenteric infarction did not develop. Paulin[11] has encountered 3

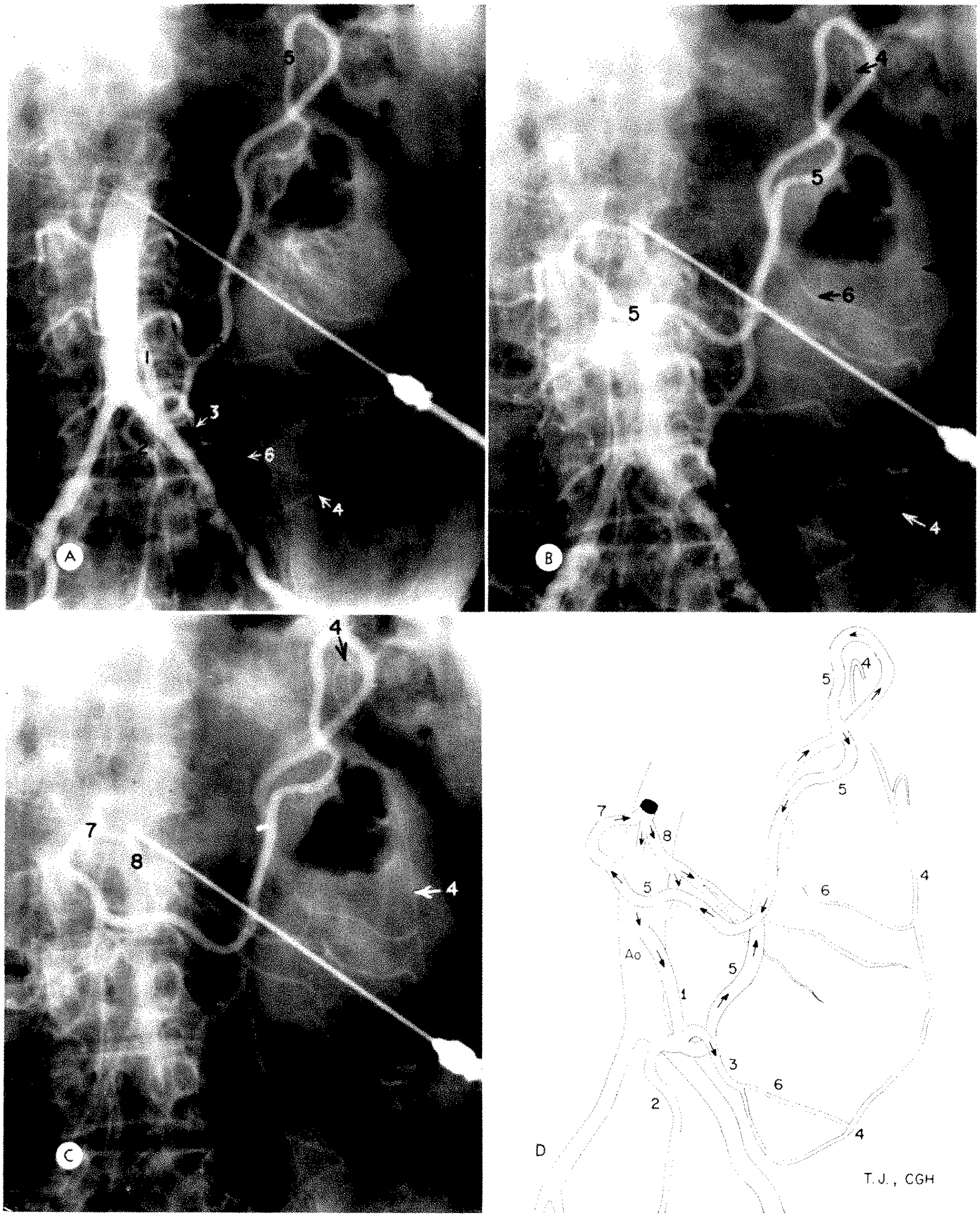

Fig. 3. (*A*, *B* and *C*) Unsuspected asymptomatic obstruction of the superior mesenteric artery. The meandering artery (5) was first recognized on the arteriogram made for evaluation of Leriche's syndrome. Note the small size of the marginal artery (4). *D* is a composite drawing of the serial examination.

surgically verified cases of celiac occlusion, 1 isolated and 2 combined with superior mesenteric occlusion. In the latter, angiograms demonstrated that the celiac and superior mesenteric arteries filled from the inferior mesenteric via a meandering artery. Kahn and Abrams[6] reported a similar case.

OCCLUSION OF THE SUPERIOR MESENTERIC ARTERY

In these cases blood reaches the superior mesenteric circulation almost entirely through the pancreaticoduodenal arcades and the anastomotic arteries of the colon. If the occlusion is at the ostium of the superior mesenteric artery, as it often is, a bridging collateral of large size is occasionally seen extending from the celiac to the main stem of the superior mesenteric, simply bypassing the obstruction. This is called Bühler's artery.[9] When it is present, there may be no need for other collaterals. We have only recognized it on one occasion.

In most cases, however, the meandering artery fills from the inferior mesenteric artery and courses proximally, parallel to the descending and distal transverse colon. It eventually terminates in the middle, or occasionally the right colic, from which it drains into the main channel of the superior mesenteric. From here the blood flows distally to supply the small bowel and proximal colon in the normal manner (Fig. 3). On 2 occasions the meandering artery bifurcated into 2 equal-sized vessels in the region of the splenic flexure. One branch, probably the accessory anastomotic artery, proceeded to the main superior mesenteric trunk (above the origin of the middle colic) while the other extended to the middle colic in 1 instance (Fig. 2, *A–D*) and to the ileocolic in the other.

If the superior mesenteric occlusion lies distal to the middle colic take-off or in the smaller divisions, the meandering artery will not be seen. Instead, bridging collaterals in the mesentery generally provide a bypass around the occlusion. In these cases the blood flow is hard to trace because of the relatively small size of the bridging vessels. We have not encountered a right-sided meandering artery in the few cases of distal occlusion in the superior mesenteric artery that we have seen.

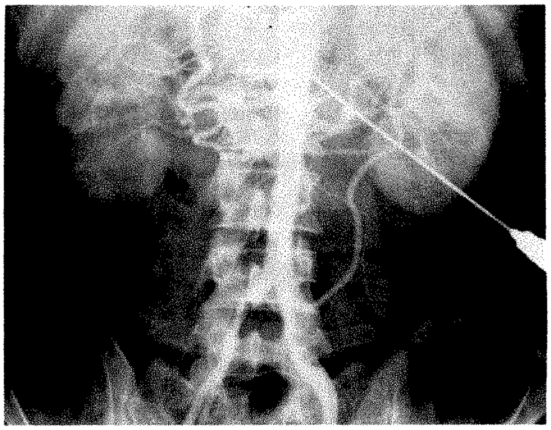

Fig. 4. Occlusion of the orifice of the superior mesenteric artery. The collateral blood is supplied through the pancreaticoduodenal arcades and the central anastomotic (meandering) artery, which fills from the inferior mesenteric.

We have reviewed 7 examples of occlusion of the main trunk of the superior mesenteric artery. In each, a typical meandering vessel (the central anastomotic artery) was clearly shown, with flow in a cephalad direction. In none of the cases could we demonstrate a continuation of this artery to the ascending colon; the ileocolic and usually the right colic filled in normal fashion after the opacified collateral blood had reached the main channel of the superior mesenteric artery (Fig. 2 through 4).

OCCLUSION OF THE INFERIOR MESENTERIC ARTERY

In 1 of our 2 cases of occlusion of the orifice of the inferior mesenteric artery, the meandering artery filled from the left division of the middle colic. It contributed branches to the descending colon, directly and through incomplete segments of the marginal artery. The meandering (central anastomotic) artery finally reached the main trunk of the inferior mesenteric, from which the sigmoid and superior rectal arteries filled in the normal direction.

In the other case an interesting variation was found. A rather large vessel arose from the left fourth lumbar artery, extended with slight undulations into the left upper quadrant, giving off branches to the descending colon. It lay medial to the usual position of the central anastomotic artery, which was not seen. The origin of the superior rectal artery was not clear. We have encountered no examples of isolated obstruction of the superior rectal, sigmoid, or other branches of the inferior mesenteric.

OCCLUSION OF THE ABDOMINAL AORTA

Occlusion of the aorta above the superior mesenteric artery, with survival, is quite rare. A recent case is reported by Lipchik *et al.*[7] Obviously this is because blood flow to both renal arteries is cut off. However, in 1 of our cases, a thrombus, extending obliquely across the aorta, obstructed the superior mesenteric and the left renal artery but permitted flow to the right renal artery (Fig. 8*C*). The patient survived this event for several years. The celiac artery and its branches were huge. The superior mesenteric distribution was supplied principally through very large pancreaticoduodenal anastomoses. The meandering artery, which had been present on an earlier study (Fig. 8, *A* and *B*) made at a time when the clot lay entirely below the renal arteries, was not present on the later study. A dilated branch from the celiac artery appeared to supply the left colon. The blood supply to the sigmoid and rectum could not be ascertained.

With complete occlusion of the aorta between the superior and inferior mesenteric artery, a rather common site, the meandering artery is always seen. It represents the central anastomotic artery and conducts blood to the main trunk of the inferior mesenteric. This, in turn, supplies the sigmoid and superior rectal arteries, the latter anastomosing with the middle and inferior rectal arteries. If the distal aorta is also filled with clot (Fig. 5), the superior rectal artery is enlarged and carries a considerable amount of the shunted blood to the hypogastric and external iliac arteries. If the lower aorta is patent, blood re-enters the aorta through the inferior mesenteric and flows distally in the common iliac arteries. Muller and Figley[10] described such a case and we have also seen one (Fig. 6, *A*, *B* and *C*). If the aortic obstruction is *confined* to the region below the origin of the inferior mesenteric, the superior rectal becomes a major pathway to the hypogastric and external iliac arteries and there is, of course, no meandering artery. As stated earlier, we have never seen the meandering artery extend down to the region of the sigmoid and rectum. Instead, it terminates in the main channel of the inferior mesenteric, from which the blood flows distally into the sigmoid and superior rectal arteries.

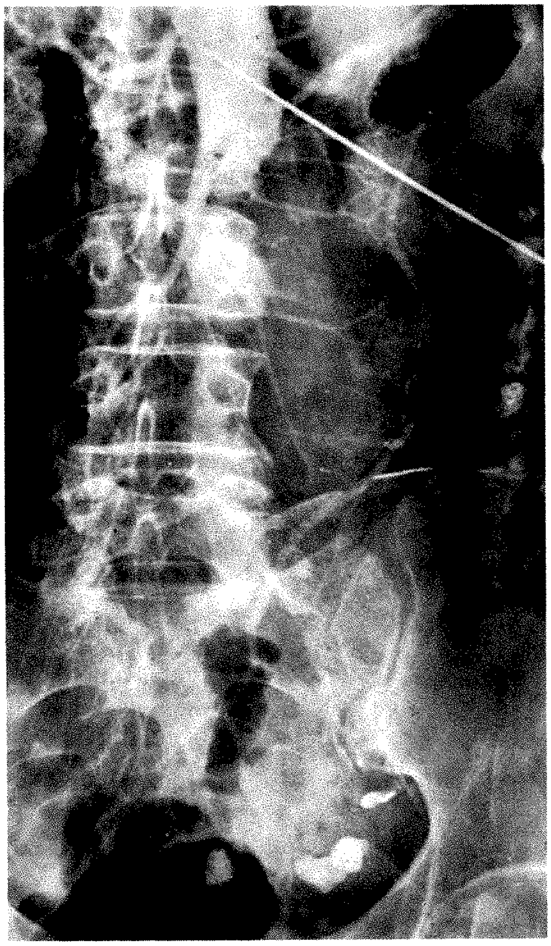

FIG. 5. Occlusion of the aorta between the superior and inferior mesenteric arteries by a clot-filled aortic aneurysm. The inferior mesenteric orifice is also blocked and the central anastomotic (meandering) artery, supplied by the middle colic, is directly continuous with the superior rectal artery. Note that the meandering artery lies at some distance from the colon, outlined by residual barium.

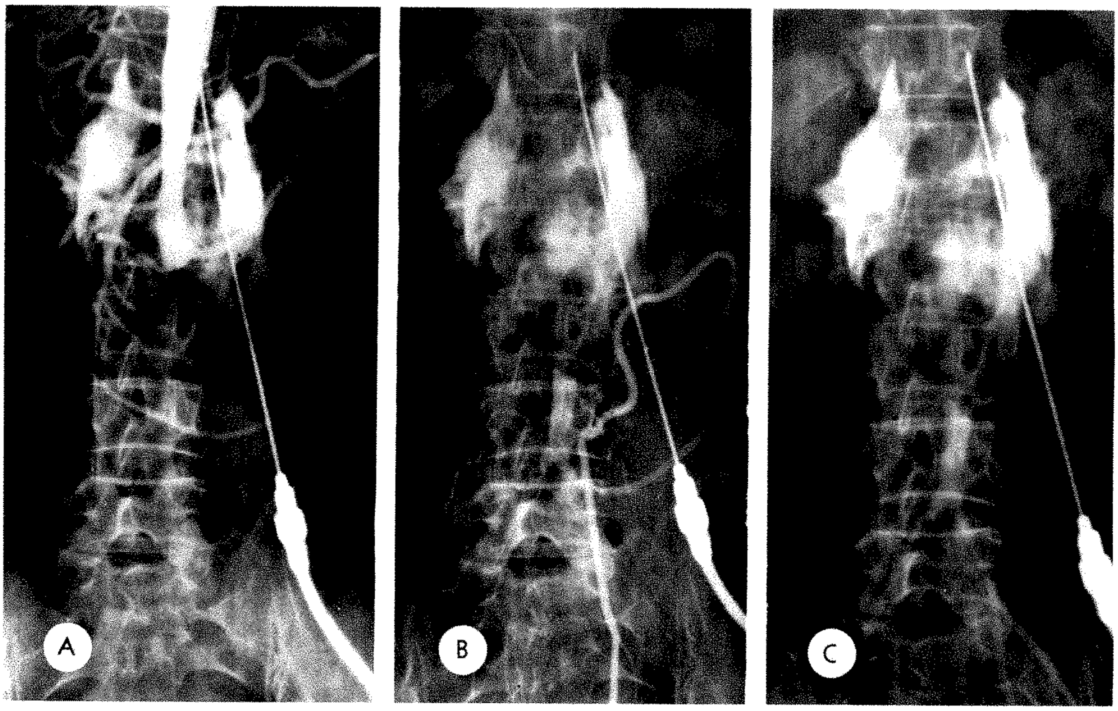

FIG. 6. (*A*, *B* and *C*) Occlusion of the aorta just distal to the renal arteries. The meandering central anastomotic artery fills from the middle colic and empties into the distal aorta via the main trunk of the inferior mesenteric artery. The superior rectal artery is enlarged. No marginal artery is seen. Considerable periaortic leakage of the contrast medium has occurred.

Of the 9 aortograms studied which demonstrated aortic occlusion between the 2 mesenteric arteries, all but one showed a meandering artery. In the exception, a thin patent channel was present along the left wall of the aortic down to the inferior mesenteric artery. Thus a pressure differential between the superior and inferior mesenteric arteries did not exist, and the anastomotic pathways did not function. Including the case with distal patency of the aorta, cited earlier, the superior rectal artery was always quite large and provided a major linkage to the hypogastric and internal iliac arteries.

In 1 patient, an abdominal arteriogram was available after the aorta was grafted. In this instance the meandering artery was no longer seen. Rogoff[17] had a similar case.

DISCUSSION

The recognition of the meandering mesenteric artery of the colon is sufficient evidence, by itself, to conclude that major abdominal arterial occlusion is present. Observing the origin and termination of this vessel and the direction of flow within it, the site of the obstruction can be accu-

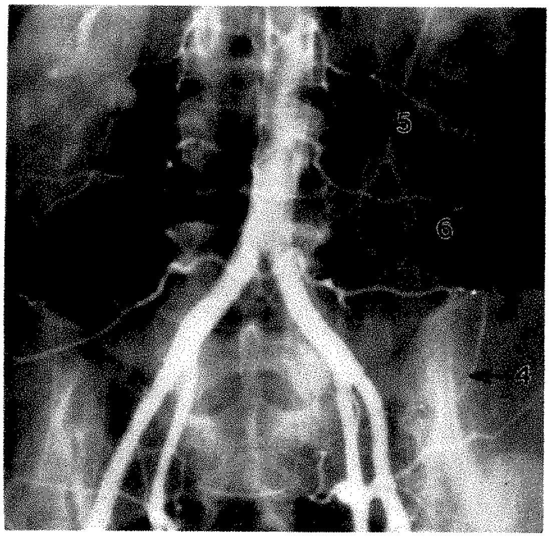

FIG. 7. A well-developed marginal artery (4) filling from a normal central anastomotic artery (5) through arterial arcades (6) in a patient who has had an abdominoperineal resection for carcinoma of the rectum.

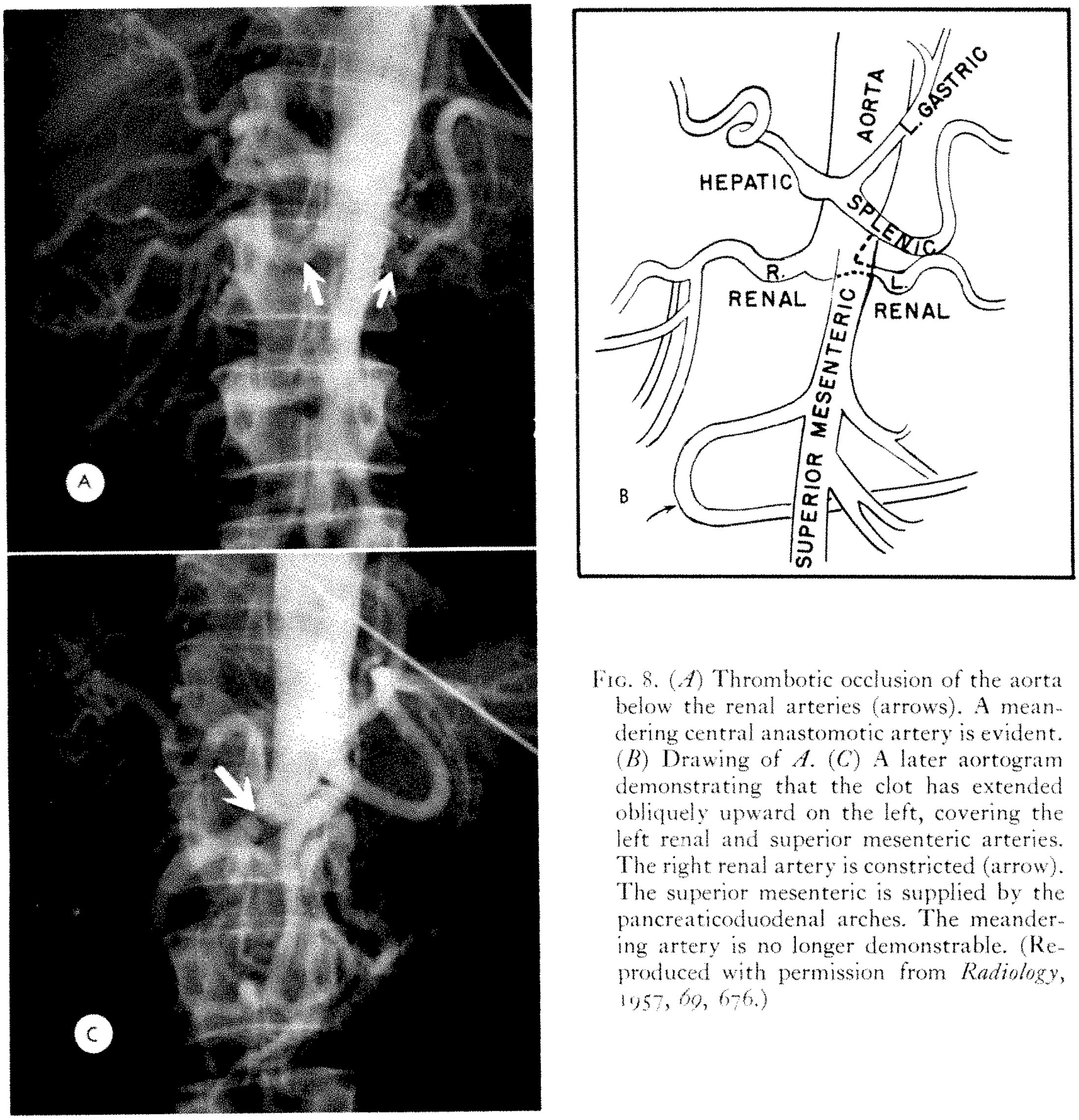

Fig. 8. (*A*) Thrombotic occlusion of the aorta below the renal arteries (arrows). A meandering central anastomotic artery is evident. (*B*) Drawing of *A*. (*C*) A later aortogram demonstrating that the clot has extended obliquely upward on the left, covering the left renal and superior mesenteric arteries. The right renal artery is constricted (arrow). The superior mesenteric is supplied by the pancreaticoduodenal arches. The meandering artery is no longer demonstrable. (Reproduced with permission from *Radiology*, 1957, *69*, 676.)

rately predicted. For example, with injection made into the lower aorta, if the meandering artery is visualized and fills from the inferior mesenteric, a diagnosis of superior mesenteric occlusion can be made. Conversely, filling of this key vessel from above indicates inferior mesenteric occlusion or aortic obstruction between the two mesenteric arteries. We have seen several instances in which the site of the occlusion of the superior mesenteric artery was not visible because of overlapping vessels or low injection, and the meandering artery, filling from the inferior mesenteric, provided the conclusive evidence of obstruction. Since the inferior mesenteric artery may not always be visualized in the normal aortogram, the presence of the meandering artery distinguishes between inferior mesenteric occlusion and technical failure. Similarly, in unsatisfactory examinations resulting from delay in film exposure, slow injection, etc., visualization of a meandering artery may still provide a clue to the diagnosis.

The clinical, surgical, and pathologic applications of knowledge of the collateral circulation to the intestines is of considerable importance. Mesenteric artery occlusion may give rise to a whole spectrum of clinical and pathologic findings. At one end of this spectrum is infarction of the intestine and at the other there is total absence of symptoms or recognizable pathologic change. In between are reversible hemorrhagic edema, ulceration, and stricture, and the symptoms and signs resulting from them, including abdominal angina. One might ask why, in the presence of such useful collateral channels as the anastomotic arteries, does infarction take place at all? The answer seems to lie in the general status of the other arteries supplying the gastrointestinal tract.[12,13] For example, in superior mesenteric artery occlusion, the celiac and inferior mesenteric arteries provide the main collateral flow. Narrowing or occlusion of these vessels sufficient to preclude adequate supportive circulation results in a varying degree of ischemia, accounting for the differences in the pathologic findings. If these sources for collateral flow are normally patent, the gut will usually remain intact. The same applies to inferior mesenteric artery occlusion which, according to Reiner *et al.*,[13,14,15] is at least as frequent as superior mesenteric occlusion.

It should be pointed out, however, that patency of the collateral vessels is not the only factor which determines infarction. The abruptness of the primary occlusion (embolism versus gradual thrombosis), its location (main stem or branches), and conditions which predispose to sluggish flow and diminished arterial pressure (polycythemia, congestive failure, etc.) all play a role.

The anastomotic arteries are also important in maintaining viability of the gut after resection of the colon (Fig. 7) and after major surgical procedures on the aorta. As a rule, if the anastomotic arteries are left intact, the intestines will remain viable. For similar reasons occlusion of the celiac axis is well tolerated.

The anastomotic arteries of the colon are among the most important vessels in the abdomen. The identification of the meandering central anastomotic artery and an appreciation of the role it plays will aid immeasurably in the recognition, understanding, and management of circulatory disease in the abdomen.

SUMMARY

We have attempted to describe the anatomy of the mesenteric circulation and to clarify its terminology. The major anastomoses between the mesenteric arteries have been pointed out, and it is emphasized that visualization of a "meandering" artery indicates the presence of an occlusive arterial process in the abdomen. Once the direction of flow in this serpentine collateral is established, the precise location of the occlusion can usually be pinpointed.

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INDICATIONS FOR SELECTIVE CELIAC AND SUPERIOR MESENTERIC ANGIOGRAPHY*

EXPERIENCE WITH 128 CASES

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SELECTIVE celiac and superior mesenteric angiography offers a radiologic assessment of the upper abdominal organs. It demonstrates the nature of disease processes involving these organs and defines the vascular anatomy (arterial and venous) important in surgical treatment.

There is no ideal radiologic method of visualizing the solid viscera of the upper abdomen. Plain roentgenograms and barium studies of the gastrointestinal lumen show only gross enlargement of the liver, spleen, or pancreas, and rarely can the etiology of the disease process be determined. The radioactive liver scan for locating hepatic tumors has been shown to be limited to demonstrating relatively large growths, at least 2.0 cm. in diameter, and occasionally it fails to detect lesions of considerably greater size.[5,8,13] Thorotrast radiographic visualization of the liver carries the hazard of inducing malignant disease in long-term survivors.[21] Splenoportography, via percutaneous splenic pulp puncture, is occasionally of value in the roentgenographic evaluation of the liver but has limitations.[1] Percutaneous cholangiography is useful principally in determining the etiology of obstructive jaundice and must usually be followed promptly by laparotomy.

Selective celiac and superior mesenteric angiography combines the advantages of some of the above techniques and is complementary to others. In addition, it demonstrates pathologic vascularity, differentiating malignant from benign masses.

In 1951, Bierman *et al.*[3] first performed selective arteriography of the celiac axis and superior mesenteric artery through brachial or carotid arteriotomy. In 1958, Ödman[15] reported 61 cases of selective celiac angiography, with introduction of the catheter through the femoral artery by the percutaneous technique of Seldinger.[20] Recently, Baum and his associates[2] have reported 75 cases, and Glenn *et al.*[7] have cited 40 cases in which the latter method was used.

The present paper records the experience with 128 consecutive cases studied by selective celiac and superior mesenteric angiography at the Massachusetts General Hospital. The more common indications for this procedure will be enumerated and analyzed, and representative examples will be presented.

METHODS AND MATERIALS

Except in 2 cases, selective celiac and superior mesenteric angiography was performed via percutaneous puncture of the femoral artery. The presence of a femoral pulse and at least one more distal pulse (popliteal, dorsalis pedis, or posterior tibial) was a prerequisite. In the 2 excepted cases, celiac and superior mesenteric catheterization was carried out by a percutaneous puncture of the brachial artery.

The only contraindications to the pro-

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cedure were the absence of pulses, as noted above, severe hemorrhagic states, and a history of reaction to iodinated contrast material. Patients with prothrombin times as low as 29 per cent, moderate anemia, or thrombocytopenia underwent the examination without complication.

The patient receives preliminary medication with demerol and nembutal and is allowed a clear liquid oral intake in order to diminish the concentration of contrast material excreted in the kidneys. The procedure is performed in the Radiology Department under local anesthesia.

The radiologist utilizes the Seldinger technique of arterial catheterization.[20] A red Kifa end-hole catheter, shaped by immersion first in boiling and subsequently in cold water, is employed. After procaine injection into the perivascular tissues, a New needle[14] is introduced into the femoral artery just below the inguinal ligament, and a guide wire is inserted through the needle into the common iliac artery. The needle is then withdrawn, leaving the wire in place, and the preformed catheter is inserted over the indwelling guide wire and passed in a retrograde manner into the upper abdominal aorta, following which the guide wire is removed. Under televised fluoroscopy, the tip of the catheter is placed in the celiac axis, and a test injection of contrast agent is made. If this is satisfactory, 30 to 50 cc. of 76 per cent renografin is injected by pressure, to deliver 8 to 10 cc./second. Single-plane serial angiograms are then obtained by an Elema Schönander changer, usually 2 per second for 8 seconds, to show the arterial and capillary phases. Late angiograms, one every third second for an additional 20 seconds, demonstrate the contrast medium draining from the viscera via the portal system. The tip of the catheter is then withdrawn from the celiac axis and introduced into the superior mesenteric artery. A pressure injection of 20 to 40 cc. of renografin is made, and multiple serial films are again exposed. Frequently, additional injections into one or both vessels are necessary, with oblique or lateral projections, to better define the disease process. Occasionally, catheters are introduced into both femoral arteries, with simultaneous catheterization and injection of the celiac and superior mesenteric artery.

ANATOMY

The vascular anatomy demonstrated by selective celiac and superior mesenteric

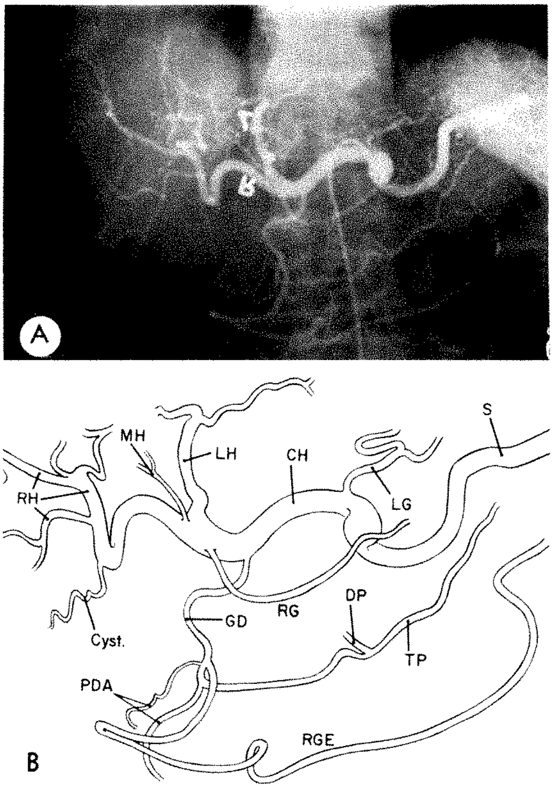

Fig. 1. Case 1. (*A*) Arterial phase of normal selective celiac angiogram. (*B*) Line drawing of *A*. The celiac axis is the first major abdominal branch of the abdominal aorta originating anteriorly at the T-12, L-1 level. It trifurcates into left gastric (LG), splenic (S), and common hepatic (CH) arteries. The common hepatic first gives off a gastroduodenal (GD) artery, then a right gastric (RG) and left (LH), middle (MH), and right hepatic (RH) arteries. The gastroduodenal artery gives branches to the head of the pancreas via the pancreaticoduodenal arcades (PDA). These arcades consist of the posterior pancreaticoduodenal artery and anterior pancreaticoduodenal artery, which in this example anastomose to form the inferior pancreaticoduodenal (IPD) artery; this vessel originates from the superior mesenteric artery. The gastroduodenal artery continues as the right gastro-epiploic artery (RGE).

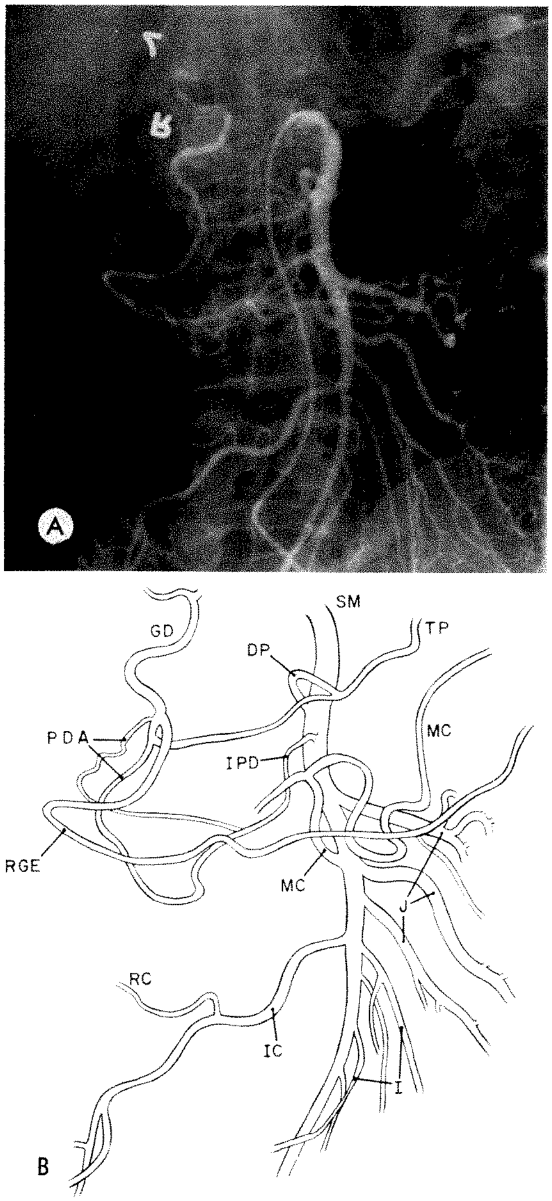

Fig. 2. Case 1. (*A*) Arterial phase of normal selective superior mesenteric angiogram. (*B*) Line drawing of *A*. The superior mesenteric artery (SM) is the second major abdominal branch of the abdominal aorta originating anteriorly at the level of L-1. The superior mesenteric artery in this case gives rise to a large dorsal pancreatic artery (DP), which in most instances, however, originates from the celiac or splenic arteries. The dorsal pancreatic artery then supplies the pancreas via the transverse pancreatic artery (TP). The next branch of the superior mesenteric artery is the common inferior pancreaticoduodenal artery (IPD) which anastomoses with the pancreaticoduodenal arcades (PDA). The superior mesenteric artery thereafter gives several jejunal branches (J) to the left and colic branches (middle—MC, right—RC, ileocolic—IC) to the right. Below the level of the ileocolic artery (IC), the superior mesenteric artery gives off numerous ileal branches (I).

angiography closely follows the pattern presented in the text by Michels.[10] Figures 1, *A* and *B* and 2, *A* and *B* illustrate the vascular distribution in a normal subject. Anomalies are extremely common, the more surgically significant being: (a) a replaced or accessory right hepatic artery arising from the superior mesenteric artery, seen in 14 per cent of cases in this series; (b) left hepatic branches originating from the left gastric artery, present in 25 per cent of a reported anatomic series;[11] and (c) absence of the middle colic artery seen in 3 per cent of an anatomic series.[12]

INDICATIONS

Indications for celiac and superior mesenteric angiography are several. They may be classified on the basis of the organ deriving its vasculature from these arteries or by the vascular system itself. Many processes may be studied, including liver or pancreatic neoplasms, pancreatitis, vascular lesions, unexplained masses, splenic trauma, and a variety of miscellaneous conditions. In addition, the procedure permits preoperative evaluation of both arterial and portal venous blood supply (Table 1).

RESULTS

Of 128 patients originally chosen for selective catheterization of the celiac and/or superior mesenteric arteries, 15 could not be catheterized and in 8 interpretation was impossible due to technical factors, leaving 105 satisfactory cases.

The median age in this study was 63 years; the oldest patient was 83 and the youngest 5. (The catheterization in the 5 year old child was performed via femoral arteriotomy.) The over-all incidence of unsuccessful attempts to catheterize the celiac and superior mesenteric artery was

Table I

indications for selective celiac and superior mesenteric angiography classified by organ or vascular system (128 patients)*

Indication	Cases Examined for This Indication	Proved Cases	Proved Cases in Which Interpretation Was Correct
I. Liver			
(a) Neoplasm	73	(See Table II)	
(b) Other liver masses	3	3	3
II. Pancreas			
(a) Neoplasm	21	13	2
(b) Pancreatitis	8	3	1
(c) Pseudocyst	2		
III. Vascular			
(a) Arterial			
occlusive	9		
preoperative evaluation	2	1	1
(b) Venous			
portal vein patency (portal hypertension)	15	5	5
patency of shunt	2	1	1
(c) Arteriovenous malformations	1	1	1
IV. Bowel			
(a) Neoplasms	7	5	4
V. Spleen			
(a) Trauma	3	2	2
VI. Miscellaneous			
(a) Unexplained mass	9	5	5
(b) Bleeding	3		
(c) Unexplained pain	1		

* Many of the cases presented with 2 or even 3 indications and may be represented under more than one category. (In cases of primary malignant disease of other organs, the liver was also examined for metastases.)

12 per cent. In the first quarter of the series, failure to obtain a successful catheterization occurred in 23 per cent of the patients; in the last quarter this figure was reduced to 6 per cent, with the age and general clinical condition of the patients remaining roughly the same. Failure was due most frequently to iliac artery tortuosity and sclerosis, prohibiting passage of the catheter or its proper manipulation (5 cases). Other causes of failure were related to anomalies or stenoses of the celiac and superior mesenteric arteries (4 cases), failure of fluoroscopic equipment (1 case), and excessive bleeding around the catheter requiring early termination of the procedure (2 cases); 3 failures were unexplained.

The only potentially serious complication occurred early in the series in a 76 year old man; a thrombosis of the femoral artery localized to the site of puncture was immediately recognized and successfully removed under local anesthesia. Two patients lost their dorsalis pedis pulses (1 for only 24 hours) without any symptomatic changes, the other pulses remaining unaffected. In 2 instances what appeared clinically to be thrombophlebitis developed within a month after the procedure. The relationship here is uncertain in that both

TABLE II

RESULTS OF THE EVALUATION OF LIVER MALIGNANCY BY SELECTIVE CELIAC AND SUPERIOR MESENTERIC ANGIOGRAPHY AS VERIFIED BY SURGERY, AUTOPSY, OR POSITIVE NEEDLE BIOPSY

Interpretation	Total Proved Cases	Interpretation Proved Correct	Interpretation Proved Incorrect	Over-all Accuracy in Proved Cases
Positive	15	13	2	86%
Negative	21	18	3	
Questionable	5			

patients were suffering from terminal cancer.

In no case was there evidence of pancreatitis, hepatitis, transverse myelitis, or bowel or renal injury as a result of the procedure. Transverse myelitis or renal injury was not to be expected, as contrast medium was not injected directly into the spinal or renal arteries.

Proof of the angiographic findings was obtained by laparotomy, autopsy, or, in the case of the liver, by positive needle biopsy; a negative needle biopsy was not accepted as valid (Table II).

The most common indication for angiography in the present series was for evaluation of possible *hepatic metastases* in patients with otherwise operable primary tumors. The cases compiled in Table II were unselected and the large majority of these patients had a current or previously known primary neoplasm outside the liver. In most of the cases with positive studies, no conclusive clinical or laboratory evidence of metastatic involvement was available prior to angiography.

Malignant disease of the liver manifests itself by angiographic patterns similar to those seen in malignant disease of the brain or meninges as shown by carotid angiography. Characteristic of liver metastases were vascular displacement, tumor staining, new vessel formation with tumor sinusoids, and filling defects in the late capillary phase (Fig. 3).

The diagnostic accuracy in the evaluation of liver metastasis by hepatic angiography was 86 per cent in proved cases. In 2 instances the findings were incorrectly regarded as positive for metastatic disease and 3 were incorrectly called negative. In 2 patients lesions were shown that could not be found at surgery, but autopsy within a short period of time showed them to be present. Metastases as small as 1 cm. in diameter were demonstrated.

In the 13 subsequently proved studies

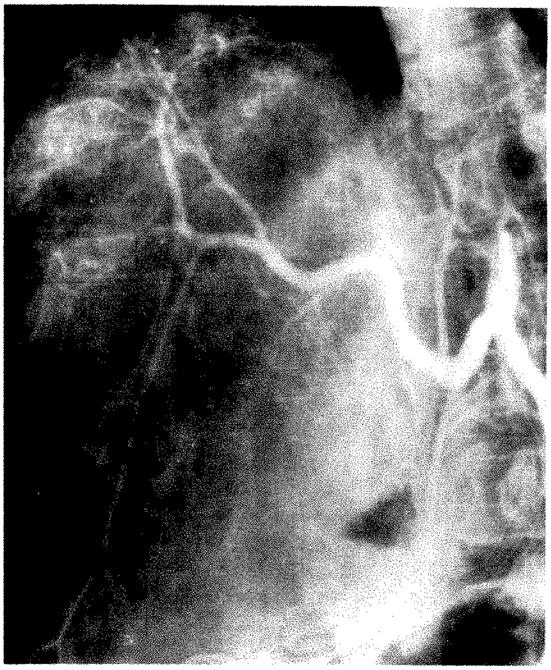

FIG. 3. Case II. This celiac angiogram was obtained in a 63 year old female with a history of carcinoma of the endometrium removed 5 years previously. At recent exploratory surgery, an enlarged but otherwise normal liver was found and liver biopsy was negative. The angiogram, made 1½ months later, shows stretching and curvilinear displacement of vessels with irregular tumor vessels and tumor capillary staining characteristic of liver metastasis. Autopsy confirmation.

performed to evaluate the *pancreas* for possible neoplasia, diagnostic accuracy was poor. Errors consisted of misinterpretation of normal findings as pathologic and failure of the angiogram to show abnormal changes when tumor was present. In 2 cases a mass lesion diagnosed as tumor proved to be localized pancreatitis (Fig. 4).

Arterial vascular assessment was quite accurate. Anomalies, stenoses, aneurysms, and arteriovenous fistulas were clearly delineated. In patients in whom the question of portal hypertension had been raised, evaluation of the portal venous system was accurate. Occasionally, the subtraction technique was utilized for better visualization of venous structures overlying the spine. Celiac axis injection of contrast agent produced a diagnostic splenoportogram in only 75 per cent of cases. With selective catheterization of the splenic artery, however, and direct injection of contrast material into that vessel, better splenoportograms were obtained, which were consistently of diagnostic quality[17] (Fig. 5, *A* and *B*).

The vascular pattern of *bowel neoplasia* was observed in a few instances, but the number of cases is small and no conclusions can be drawn.

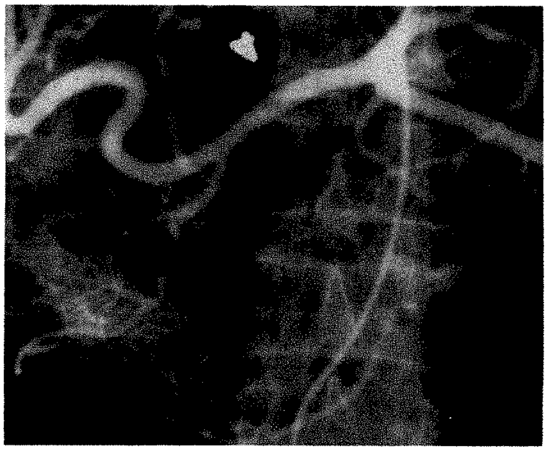

Fig. 4. Case iii. A celiac angiogram in a 77 year old female with pulmonary nodules and a normal upper gastrointestinal series shows stretching of the pancreaticoduodenal arcades indicating a primary tumor of the pancreas. No conclusive tumor neovascularization is seen. Autopsy confirmation.

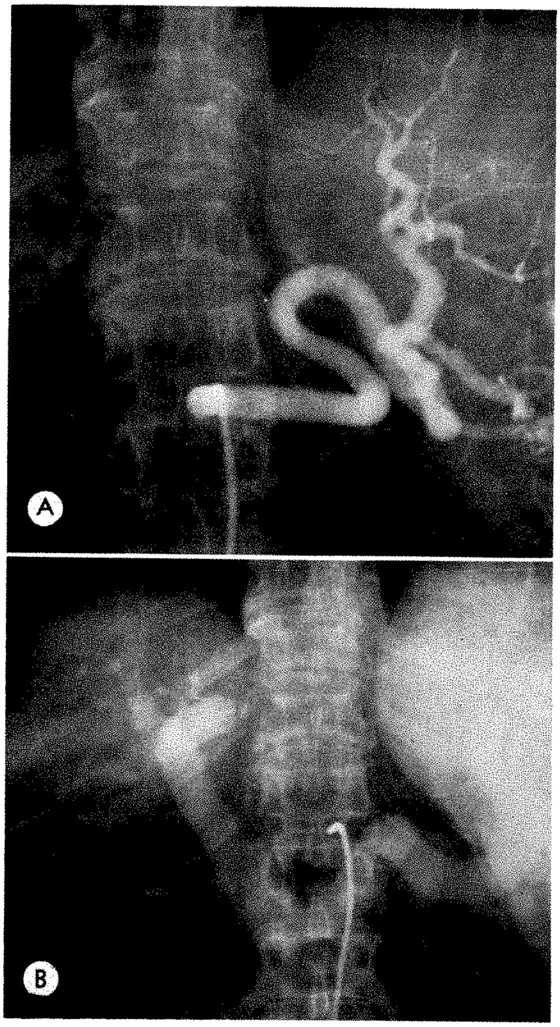

Fig. 5. Case iv. (*A*) Arterial phase of selective splenic angiogram, performed in a 68 year old woman to evaluate portal hypertension, shows an enlarged spleen. (*B*) Venous phase shows the large patent portal vein. The original films showed reflux into the short gastric and inferior mesenteric veins, corroborating portal hypertension.

Rupture of the spleen was accurately diagnosed in 2 patients by displacement of vessels, disruption of the splenic outline, loss of uniformity of contrast filling in the splenic pulp, as well as by splenic displacement due to perisplenic hematoma (Fig. 6, *A* and *B*).

Selective celiac and superior mesenteric angiography has been of help in the evaluation of *abdominal masses of unexplained etiology*. The origin of the mass may be shown by tracing its vascular supply, and its nature can often be detected by typical vascular changes, as in a malignant tumor (Fig. 7, *A* and *B*).

The procedure was not helpful in 2 patients with *unexplained gastrointestinal bleeding.*

DISCUSSION

The advantages of selective arterial catheterization over aortography are several. Aortography suffers from confusing shadows of overlying vessels as well as poor concentration in the vessels of interest. In selective arterial catheterization only the vessels of interest are filled with contrast agent, the picture is not confused by overlying vessels, and the vessel from which the lesion derives its blood supply is clearly demonstrated. Thus, the organ involved and the nature of the pathologic process are often well defined. Since the entire injection is into a smaller vessel, a good concentration of contrast material is obtained with a smaller quantity of the agent, giving a clearer and more precise delineation of abnormality. There is better filling of tumor vessels, making possible more accurate diagnosis of tumors. Since an undiluted bolus of contrast material can be rapidly delivered into the selectively catheterized artery, venous structures are often adequately demonstrated after the contrast agent passes through the viscera.

Selective celiac and superior mesenteric angiography appears particularly promising in the evaluation of liver metastases. Although a larger series will be necessary for full evaluation of this method, preliminary data have shown an over-all accuracy of 86 per cent in 36 proved cases. The procedure appears to be more accurate than percutaneous needle biopsy of the liver.[6] In this small series it has been equally or more accurate than radioisotopic liver scanning[8,13] in detecting intrahepatic mass lesions and has the advantage of differentiating benign from malignant tumors. Accurate demonstration of the nature of a mass in the liver, along with the vascular

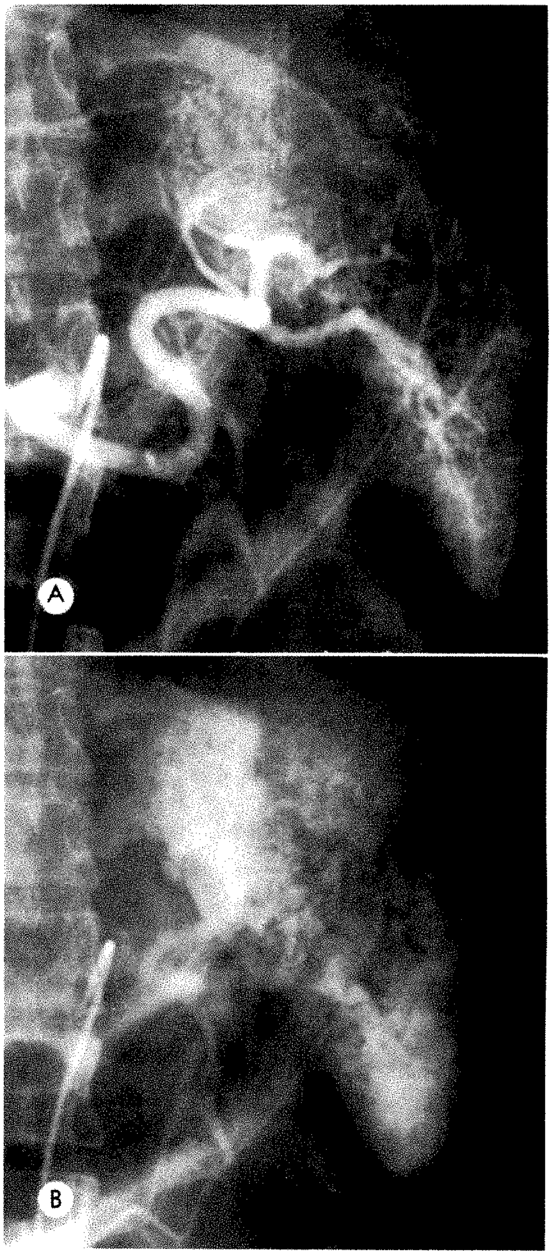

Fig. 6. Case v. (*A*) Early arterial phase of celiac angiogram in a 20 year old woman with suspected splenic rupture in whom vital signs were stable, abdominal roentgenograms were nondiagnostic, and repeated abdominal taps for blood were negative. (*B*) Later film shows rupture of the splenic capsule with a wedge-shaped filling defect, indicating an intrasplenic hematoma. The spleen is displaced from the lateral abdominal wall and diaphragm by perisplenic hematoma. Surgical confirmation.

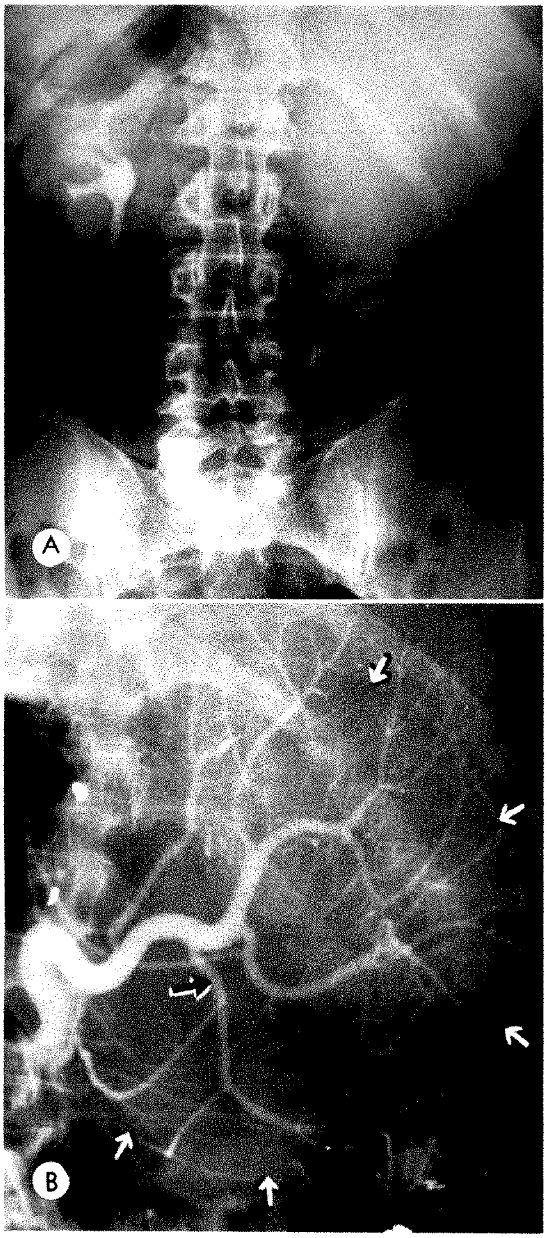

Fig. 7. Case vi. (*A*) Intravenous pyelogram in a 51 year old male, who was referred for possible hepatic lobectomy, because a tumor, thought to be confined to the left lobe of the liver, was found at laparotomy at another hospital. Note the left upper quadrant mass. (*B*) Celiac angiogram demonstrates the left gastro-epiploic artery (black arrow) and its branches encircling a large mass (white arrows). The liver was shown to be normal on other films of the angiography (not shown). Operation revealed a leiomyosarcoma of the greater curvature of the stomach and a normal liver.

anatomy, can prove helpful in the surgical planning of hepatic resection. Patients with demonstrated hepatic metastases may be spared extensive cancer resections, or a solitary liver lesion might be attacked surgically. In some patients the radiotherapist was guided by the angiogram in planning treatment fields. In others, perfusion of the liver with chemotherapeutic agents was accomplished via the catheter in the celiac axis or hepatic artery.

The disappointing preliminary results in the small series of pancreatic cancer cases may be attributable in part to the poor opacification of the pancreatic vessels due to the dual blood supply from both celiac and superior mesenteric arteries. The variable vascularity of pancreatic neoplasms appears also to play a role. Most adenocarcinomas of the pancreas are roentgenographically avascular; thus tumor blush or tumor vessels are not often seen. The variation in normal anatomy makes reliance on minor degrees of vessel displacement as a sign of neoplasia hazardous. A simultaneous superior mesenteric and celiac axis injection, as suggested by Boijsen,[4] was used in the latter part of this series without improvement in diagnostic accuracy. It had not been used earlier in the study because of the slightly increased risk assumed to be associated with puncture of both femoral arteries.

In view of their experience and the above-mentioned considerations of pancreatic and pancreatic tumor vascularity, the authors conclude that angiography of the pancreas will not prove to be a satisfactory method for the early diagnosis of disease in that occult organ. It would seem, as suggested by Potsaid,[19] that the solution of that problem lies in an as yet undiscovered iodinated substance that will concentrate within the pancreas.

Note: The markings seen along the lumbar spine in some of the illustrations are used as an aid for catheter placement.

Selective angiography is helpful in preoperative evaluation of vascular anatomy, since anomalies of the celiac and superior mesenteric branches are common. The blood supply of the colon may be assessed prior to colo-esophageal bypass. Likewise, an anomalous right hepatic artery originating from the superior mesenteric artery may be detected preoperatively and spared during a pancreaticoduodenal resection. Knowledge of the distribution of the hepatic blood supply prior to hepatectomy is valuable. The study to localize the tumor mass also aids in ruling out other hepatic masses.

Selective superior mesenteric angiography promises to be an ideal method for investigating mesenteric angina and mesenteric vascular occlusion. The arterial tree is well demonstrated in embolism and atheromatous disease, though a supplemental lateral aortogram is usually necessary, as the most frequent site of atheromata is at the origin of the superior mesenteric artery. Aneurysms of branches of this artery and other vascular malformations were conclusively demonstrated.

The portal venous system can be shown in the late phase of celiac or superior mesenteric angiography as the contrast agent returns from the spleen or bowel. As an alternative to splenic pulp puncture, this method has been helpful in the evaluation of portal vein patency and portacaval shunt patency, and in the demonstration of esophageal varices in patients with portal hypertension. A selective splenic artery injection improves the diagnostic quality of the splenoportogram.

In patients with persistent portal hypertension in whom prior splenectomy has been performed, late roentgenograms following a superior mesenteric artery injection demonstrate the portal system as the contrast agent returns from the bowel via the superior mesenteric vein. In these cases a superior mesenteric angiogram is the only practical method of preoperative demonstration of patency of the portal vein.[17]

Neoplasms of the stomach, small bowel, and large bowel have been shown. Angiographic study of these lesions, though still in the investigative stages, may perhaps prove useful in differential diagnosis. Margulis and Heinbecker[9] have shown, on occasion, differing appearances between tumors and inflammatory lesions of the bowel by selective arteriography at the operating table.

In traumatic cases selective celiac and superior mesenteric angiography holds promise, although its clinical application may be limited. The precarious state of the patient and the necessity of early diagnostic and therapeutic laparotomy often preclude the study. Splenic trauma has been accurately assessed by this method.[18] At the same examination, one may easily catheterize selectively the vessels of other solid abdominal viscera, ruling out injury to these organs. Renal trauma has been shown by selective angiography.[16]

The demonstration of the origin of abdominal masses allows preoperative planning of surgical procedures. Detection of gastrointestinal bleeding sites has been suggested,[2] but has not proved practical in most patients. Active bleeding is necessary at the time of the angiographic study in order to demonstrate the site of hemorrhage, and often the patient is in too critical a condition at such a time to justify angiography.

SUMMARY

Selective celiac and superior mesenteric angiography was undertaken in 128 instances without serious sequelae. The series included liver and pancreatic neoplasms, pancreatitis, vascular lesions, unexplained upper abdominal masses, splenic trauma, and a small group of miscellaneous conditions.

In the evaluation of suspected hepatic neoplasm, an accuracy of 86 per cent was shown in 36 proved cases. The method proved to be of little value in the study of suspected pancreatic neoplasia and was frequently misleading. It offers a unique method of demonstrating the intra-ab-

dominal vasculature, both arterial and portal venous. The procedure was useful in the evaluation of unexplained abdominal masses. Splenic rupture was successfully demonstrated.

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GASTROINTESTINAL MANIFESTATIONS OF ABDOMINAL AORTIC ANEURYSMS*

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ALTHOUGH there are numerous articles dealing with the gastrointestinal problems of patients with abdominal aortic aneurysms,[2,4,13–16,20,23,24,26,27,30,36,40,41–44,49,51,53,54,56,59,60] there is no comprehensive review of the subject. For this reason the literature was reviewed and the gastrointestinal findings of 100 patients with abdominal aortic aneurysms were evaluated. Visceral symptoms and abnormal gastrointestinal roentgen findings were encountered more frequently than heretofore recognized. The roentgenographically demonstrable abnormalities of the intestinal tract produced by aneurysms have been discussed in some of the articles,[7,19,22,25,27,32,39,58] but have been neglected in the recent literature. The purpose of the present authors is to describe these deformities, to emphasize their importance in diagnosis of abdominal aneurysms, and to discuss the differential diagnosis of abdominal masses.

MATERIALS AND METHOD

The first 100 consecutive cases of abdominal aortic aneurysms diagnosed by intravenous abdominal aortography[57] beginning in 1959, were studied for gastrointestinal involvement. Clinical records were critically evaluated for gastrointestinal complaints and abdominal findings. Symptoms referable to pre-existing or coexisting gastrointestinal disease were excluded. Conventional abdominal roentgenograms and gastrointestinal barium studies were reviewed for abnormalities resulting from the aneurysms and for evidence of gastrointestinal disease.

RESULTS

The ages of the patients ranged from 48 to 79 years; 53 were in the seventh decade and 75 were over 60 years old. The ratio of males to females was 5:1. All but one of the aneurysms were arteriosclerotic.

Fifty-five per cent of the patients in this series had abdominal symptoms (Table I). Of these, dull or colicky pain in one of the abdominal quadrants was the most common. However, this was most frequent in the epigastrium and left lower quadrant and was almost universally unrelated to meals. Aggravation of pain in the supine position was elicited in only 2 patients, and only 1 patient had a history of abdominal angina. Unexplained loss of weight was reported by 18 patients. Constipation of recent onset occurred in 10 patients, or 20 per cent of those with gastrointestinal symptoms.

TABLE I

GASTROINTESTINAL SYMPTOMS IN 100 CONSECUTIVE CASES OF ABDOMINAL AORTIC ANEURYSMS

Total no. of patients with complaints		55
Abdominal pain		24
epigastric	8	
left lower quadrant	6	
colicky lower abdominal	3	
periumbilical	2	
other areas	5	
Weight loss		18
Constipation		10
Postprandial fullness		8
Abdominal pulsations		7
Abdominal mass		6
Diarrhea		4
Anorexia		3
Indigestion		2

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Diarrhea was described by 4 patients and in 2 there was alternating constipation. Eight patients complained of postprandial fullness; nausea and vomiting occurred in 4 patients. In 1 of these, retroperitoneal rupture of the aneurysm had occurred. The other patient with rupture of an aneurysm had diarrhea and melena. Abdominal pulsations and the awareness of an abdominal mass were described by 7 patients. The incidence of associated peptic ulcer disease was 20 per cent, while gallbladder disease was present in 6 per cent of the patients. A pulsatile mass was palpated in 59 per cent of the patients, whereas a nonpulsatile lesion was present in 7 per cent. Two patients with tenderness of the abdominal mass had unruptured aneurysms.

Fifty-seven patients had conventional abdominal roentgenograms prior to intravenous abdominal aortography. Of these, the diagnosis of abdominal aortic aneurysm was evident in 58 per cent, and equivocal in 12 per cent. Only 22 of the patients in the series of 100 cases had gastrointestinal roentgenographic studies within a 5 year period prior to diagnosis of the aneurysm. Abnormalities were found in 7 (approximately 30 per cent). Distortion of the colon was found in only 1 of 17 patients who had barium enema examinations. Many of the aneurysms visualized in this series were large and most extended to the left of the aorta.

ILLUSTRATIVE CASES

Case i. A 57 year old man (N.Y.H. No. 909892) was found to have an abdominal aortic aneurysm during a routine physical examination 2½ years prior to admission. He remained asymptomatic except for weight loss (13 kg.) and was referred for surgery because of progressive enlargement of the aneurysm. Physical examination disclosed a large pulsatile mass with an audible bruit in the left side of the abdomen. Frontal and lateral roentgenograms of the abdomen revealed a curvilinear calcification which extended 10 cm. laterally and 7 cm. anteriorly to the third lumbar vertebra. The medial aspect of the mid-descending colon was indented by the aneurysm (Fig. 1*A*). An upper gastrointestinal series showed spreading of small bowel loops in the left upper abdomen (Fig. 1*B*). An intravenous abdominal aortogram disclosed a very large aortic aneurysm measuring 8 by 20 cm. in its opacified diameter with a 4 cm. thrombus along the left wall (Fig. 1*C*). At surgical exploration, an unresectable aneurysm extending from the diaphragm into both common iliac arteries was found; the abdominal viscera were normal.

Case ii. A 78 year old woman (N.Y.H. No. 900385) with a history of chronic constipation developed diarrhea 1½ months prior to admission. Loose stools alternated with constipation every 2 to 3 days. Left-sided abdominal pain, occasionally radiating to the chest and shoulder, was unrelated to meals but was associated with anorexia and weight loss (16 lb.). A hard, pulsatile, left abdominal mass, over which a bruit was heard, was palpable. Roentgen examination revealed an extrinsic filling defect of the greater curvature of the body of the stomach, elevation of the transverse duodenum, and lateral displacement of the proximal jejunum (Fig. 2*A*). A 7 by 13 cm. aneurysm lying to the left of the spine and severe right hydronephrosis were demonstrated by intravenous aortography (Fig. 2*B*). Surgery was refused by the patient; she was still alive 2 years later.

Case iii. A 63 year old white male (N.Y.H. No. 881178) with recurrent thrombophlebitis of the lower extremities was treated with anticoagulants for a 6 month period prior to admission. Persistent back pain and low grade fever followed. He also complained of constipation, which antedated the discovery of a nontender, 10 by 15 cm. pulsatile, midabdominal mass by 2 to 3 months. Gastric deformity, as well as elevation and pressure deformity of the third part of the duodenum was evident (Fig. 3, *A* and *B*). Abdominal aortography revealed a huge aneurysm extending to the right of the spine (Fig. 3*C*). At operation, a 10 by 15 cm. aneurysm beginning 1 cm. below the renal arteries and containing up to 5 cm. of thrombus was found. It was adherent to the posterior wall of the duodenum and had eroded through all layers except the mucosa. The posterior wall of the aneurysm had also eroded contiguous vertebrae andparaspinal muscles. The aneurysm was resected and replaced by a teflon prosthesis. The patient was

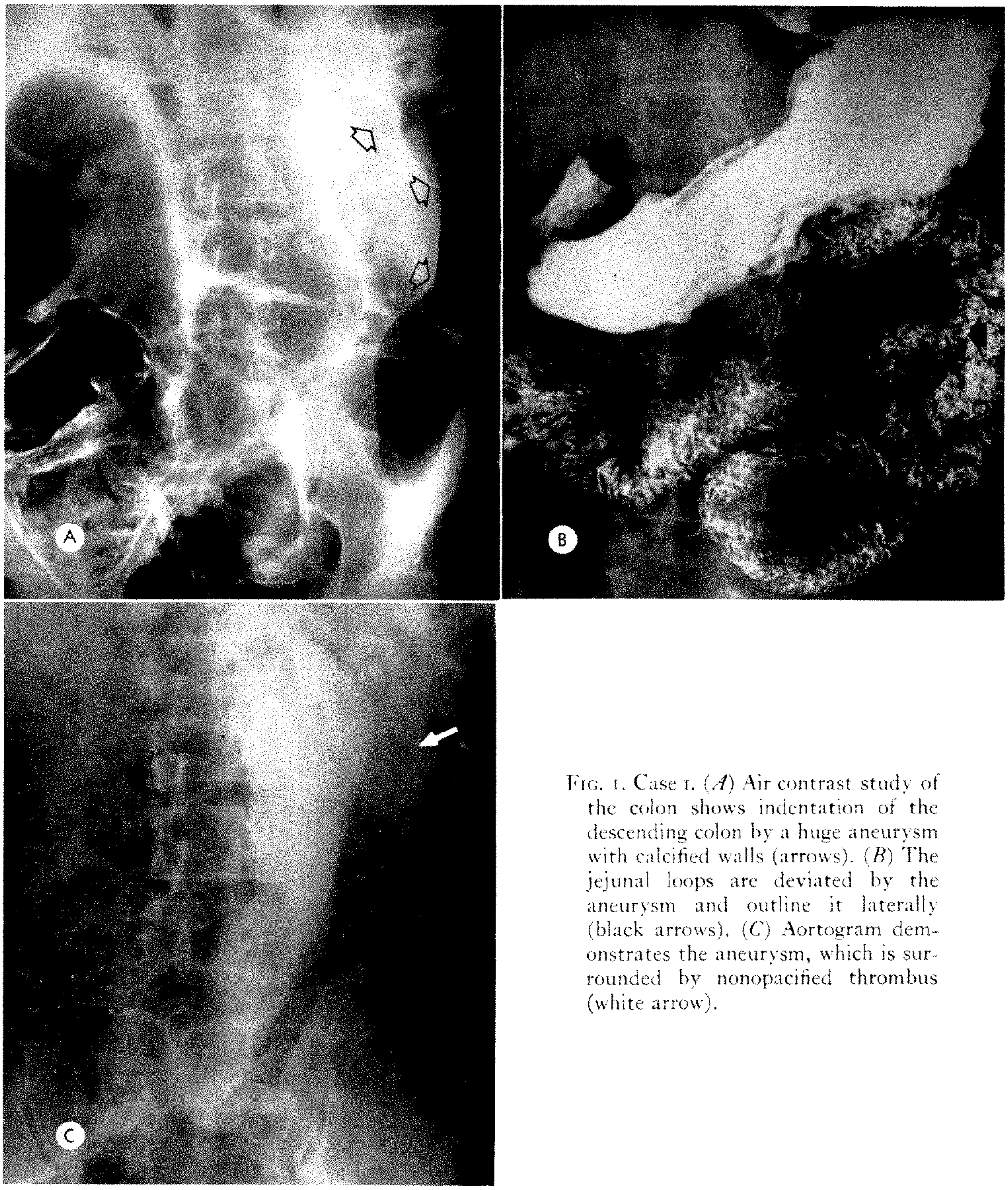

FIG. 1. Case I. (*A*) Air contrast study of the colon shows indentation of the descending colon by a huge aneurysm with calcified walls (arrows). (*B*) The jejunal loops are deviated by the aneurysm and outline it laterally (black arrows). (*C*) Aortogram demonstrates the aneurysm, which is surrounded by nonopacified thrombus (white arrow).

well for 2 years and then developed thrombosis of the left arm of the prosthesis; this was excised and replaced by a new limb prosthesis, and he improved.

CASE IV. A 65 year old hypertensive woman (N.Y.H. No. 576089) with a 27 year history of gallbladder disease was admitted because deformity of the gastric antrum had been demonstrated on a gastrointestinal series. Three years previously cholecystostomy had been performed for chronic cholecystitis, cholelithiasis, and subhepatic abscess. For the past 6 months she had complained of anorexia, epigastric bloating, and a 9 pound weight loss. Because of the roentgenographic findings, and even though a pulsatile mass was not palpated, aortography was ordered. It revealed a 4 by 7.5

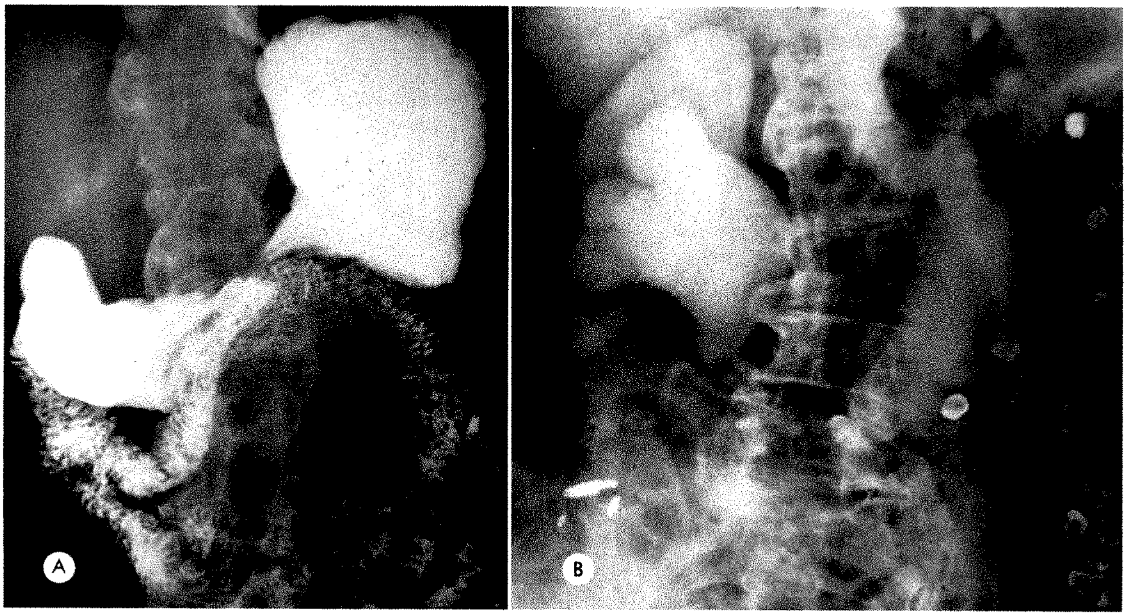

FIG. 2. Case II. (*A*) Gastrointestinal study shows the stomach draped over an aneurysm which produces a large filling defect in the greater curvature. The transverse duodenum is markedly elevated and the jejunum is deviated laterally. (*B*) Abdominal aortogram reveals a buckled aorta with a large saccular aneurysm. Advanced right hydronephrosis and multiple diverticula of the colon are also apparent.

cm. aneurysm with markedly tortuous aortoiliac and iliofemoral arteries (Fig. 4*B*). Cholecystectomy was performed 10 months later; operation disclosed that the aneurysm extended above the renal arteries and displaced the pancreas and common bile duct (Fig. 4*A*), and so it was not resected. Subsequently, the patient became asymptomatic and has remained well for 1½ years.

CASE V. A 62 year old man (N.Y.H. No. 866641) was admitted with a 2 month history of anorexia, 25 kg. weight loss, epigastric pain after eating solid foods, and dull left upper quadrant and flank pain. The latter radiated to the back and was aggravated by lying on the left side but was unrelated to meals and exercise. Physical examination revealed a pulsatile mass in the right hypochondrium. An intravenous aortogram showed a 6.5 by 10 cm. aneurysm over, and slightly to the right, of the lumbar spine (Fig. 5*C*). Narrowing of the duodenal loop, with elevation and anterior displacement of the third part of the duodenum, and a carcinoma of the distal esophagus were also demonstrated (Fig. 5, *A* and *B*). The esophageal disease was initially controlled with Co^{60} irradiation but the patient succumbed within 1½ years. Postmortem examination revealed an ulcerating carcinoma of the esophagus with extension to the stomach and a large unruptured aneurysm of the abdominal aorta displacing, but not penetrating, the duodenum.

DISCUSSION

The association of gastrointestinal disease and abdominal aneurysms was first suggested in 1806 by Laennec,[36] who described a 32 year old man who had died of a ruptured thoracoabdominal aneurysm. Autopsy disclosed engorgement of the mesenteric lymphatics which Laennec attributed to obstruction of the thoracic duct by the aneurysm. Rupture of an aneurysm into the intestinal tract was first mentioned in the lectures of Sir Astley Cooper, delivered at St. Thomas' Hospital in 1817–1818. These were recorded in notes taken by John M. Sterling and bound in manuscript form in 1822.[10] He wrote: "Aneurisms . . . may burst into the intestine, as in this specimen. The patient from whom this was taken, had a pulsatory motion in the abdomen, from which he suffered very little. At length, however, he was seized with hemor-

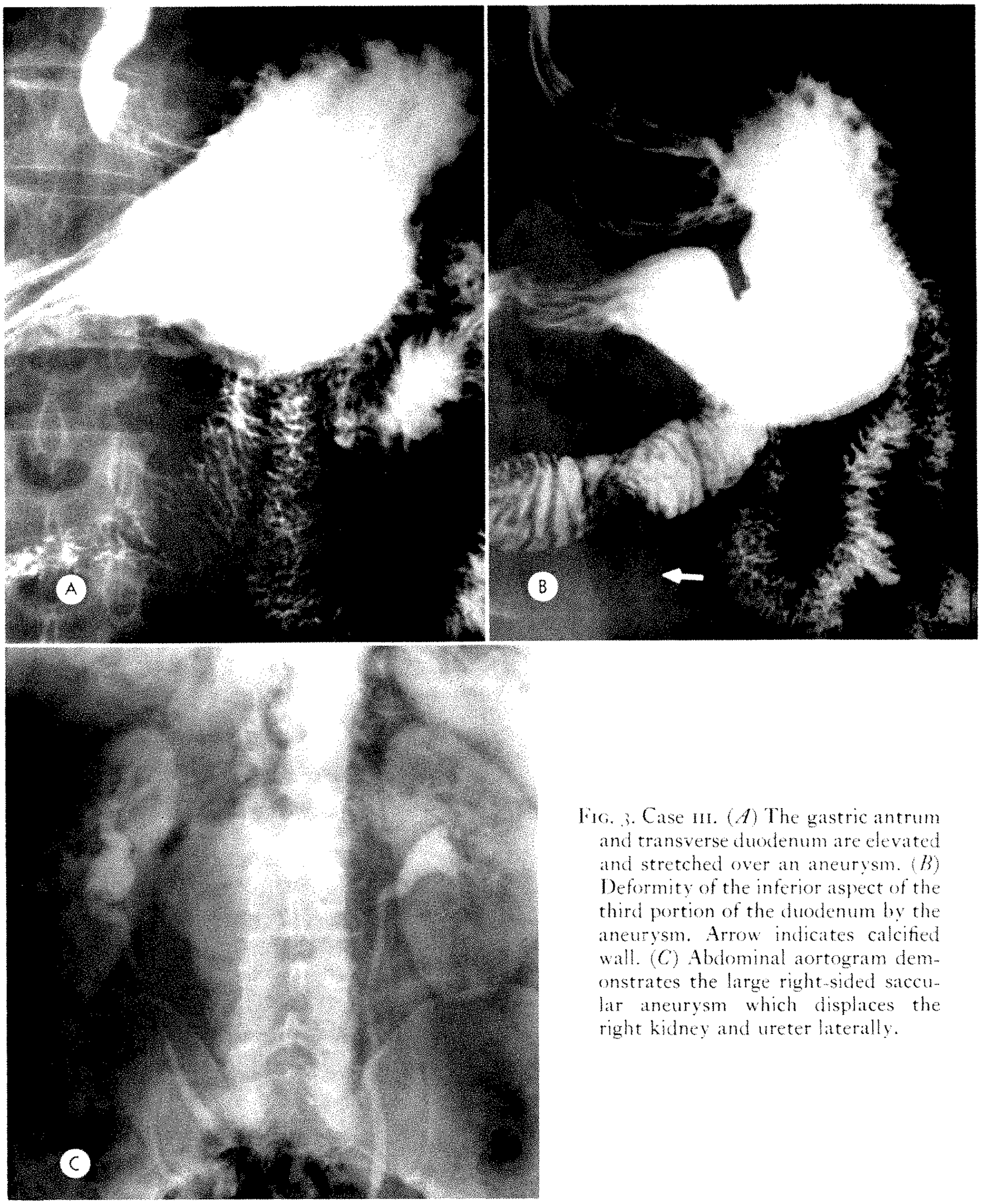

FIG. 3. Case III. (*A*) The gastric antrum and transverse duodenum are elevated and stretched over an aneurysm. (*B*) Deformity of the inferior aspect of the third portion of the duodenum by the aneurysm. Arrow indicates calcified wall. (*C*) Abdominal aortogram demonstrates the large right-sided saccular aneurysm which displaces the right kidney and ureter laterally.

rhage from the intestine, in consequence of which he fainted, recovering a little after a few hours; but in a short time he died. It was found that the aneurism had burst into the jejunum which passed over it." When these notes were published in 1825, Cooper[11] felt that abdominal aneurysms "sometimes" burst into the intestine and cited the same case.

In 1833, Chomel and Dalmas[9] were the next to discuss the subject and were also the first to offer an explanation for the

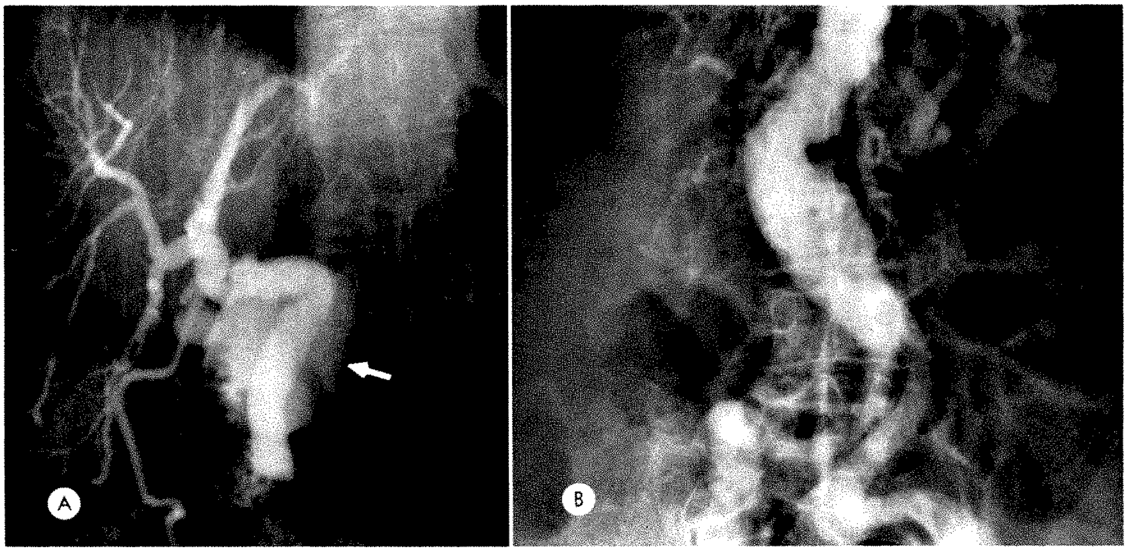

Fig. 4. Case iv. (*A*) Operative cholangiogram shows lateral deviation of the common duct by an aneurysm. The duodenal bulb (arrow) has been filled with contrast medium by retrograde peristalsis. (*B*) Intravenous aortogram demonstrates the fusiform aortic and bilateral common iliac aneurysms.

small initial volume of the hemorrhages and delayed demise of these patients, which is still valid today. They noted that the channel between the aneurysm and gut was narrow and surrounded by adhesions and fibrinous thrombus. In the most recent description of the mechanism of rupture, Garrett *et al.*[26] added that contractility of the bowel wall also restricted the hemorrhage. In 1843, Salmon[51] published the case report usually cited as the first; subsequently, about 100 cases have been reported. Rottino,[50] Hirst and Affeldt,[30] Skromak *et al.*,[53] Voyles and Moretz,[59] and Foster and Vetto[24] have published important reviews. Rupture into the third portion of the duodenum occurred in approximately 60 per cent of the cases. This is explained by fixation of the retroperitoneal portion of the duodenum between the superior mesenteric artery and the aneurysm which allows erosion. Eight cases of rupture of aneurysms into the stomach, 5 into the jejunum, 3 into the ileum, and 2 into the colon have also been reported.[24,40]

Up to 1962, DeWeese and Fry[14] collected 44 cases of rupture into the intestinal tract of homograft or plastic prostheses used in the replacement of aneurysms and suggested a method of preventing this complication. Garrett *et al.*[26] reported 12 additional cases of secondary aortoduodenal fistulas which occurred with homografts and all types of synthetic grafts. Rupture was into the terminal duodenum in all except 11 cases where it occurred into the jejunum and ileum.[14,26]

Voyles and Moretz[59] reported the first case of rupture of an aneurysm into the intestine with antemortem diagnosis and surgical repair, and re-emphasized that there is frequently a latent period between the onset of hemorrhage and death. This interval was more than 24 hours in 46 per cent of the cases and less than 6 hours in only 27 per cent. Other reports[4,24,26] have substantiated this experience. With timely diagnosis remedial surgery is, therefore, possible and has permitted survival in several cases.[40,41,49]

An unusual gastrointestinal complication of abdominal aneurysms is duodenal obstruction. Only 8 cases are cited in the world literature, and these have not been previously reviewed. Although Spisharny[56] is credited with the first case report, Osler[48] described a case 2 years earlier. His patient, an elderly woman with severe abdominal

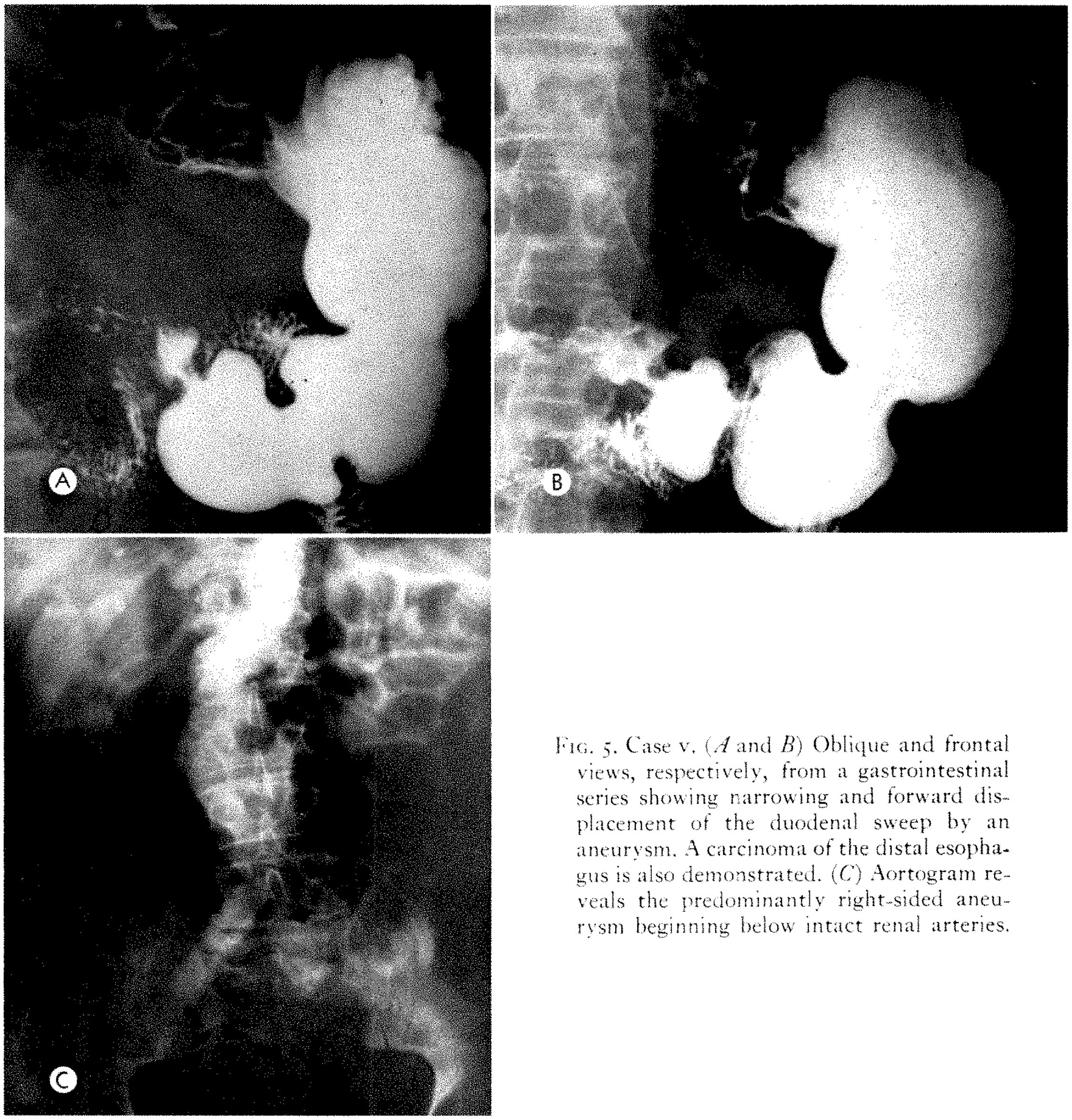

Fig. 5. Case v. (*A* and *B*) Oblique and frontal views, respectively, from a gastrointestinal series showing narrowing and forward displacement of the duodenal sweep by an aneurysm. A carcinoma of the distal esophagus is also demonstrated. (*C*) Aortogram reveals the predominantly right-sided aneurysm beginning below intact renal arteries.

pain and distention, was found to have an enormously dilated stomach due to pressure of an aneurysm on the duodenum. The patient described by Spisharny[56] in 1907, had a history of intractable nausea and vomiting, abdominal pain, and emaciation. He had an aneurysm at the level of the celiac artery which had obstructed the duodenum at the ligament of Treitz. Washburn and Wilbur[60] published the first roentgenogram of such a case, showing the dilatation of the duodenal sweep and pressure deformity of the third portion of the duodenum. His patient, a 63 year old woman with a 4 year history of weight loss, epigastric distress, and intermittent vomiting had a 7.5 cm. aneurysm which had caused dilatation and hypertrophy of the duodenum proximal to the site of obstruction. Dunning and Jones[16] described a 76 year old man with a 3 month history of 50 pound weight loss and vomiting. A barium meal study revealed dilatation of the second, and proximal portion of the third

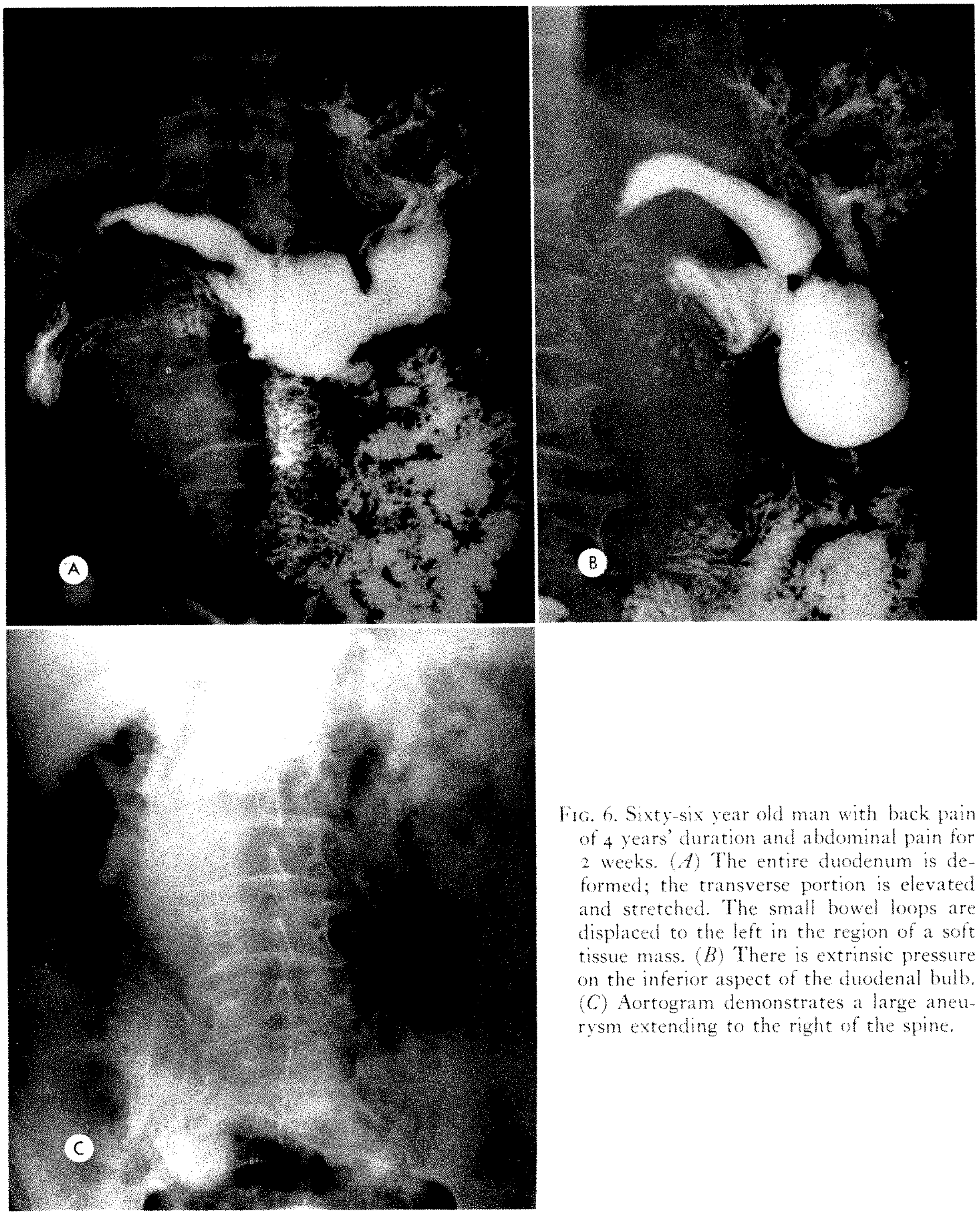

Fig. 6. Sixty-six year old man with back pain of 4 years' duration and abdominal pain for 2 weeks. (*A*) The entire duodenum is deformed; the transverse portion is elevated and stretched. The small bowel loops are displaced to the left in the region of a soft tissue mass. (*B*) There is extrinsic pressure on the inferior aspect of the duodenal bulb. (*C*) Aortogram demonstrates a large aneurysm extending to the right of the spine.

duodenum and delay of the meal in this region. A 5 by 7 cm. aneurysm extending upward from the origin of the inferior mesenteric artery was found. Javid *et al.*[34] mention 2 patients with 4 and 9 day histories of vomiting. Surgery disclosed compression of the transverse duodenum by aneurysms, one of which had ruptured prior to operation. Another 76 year old man with nausea and bilious vomiting was described by Margoles and Olsen.[42] A 9 cm. aneurysm was found compressing the third part of the duodenum and had caused proximal duodenal dilatation.

Eudel and Péquignot[20] described a 62 year old man with an 11 year history of epigastric pain. Weight loss, recurrent severe abdominal pain, nausea, and vomiting were of four years' duration. Laparotomy revealed a "voluminous antral tumor" which was adherent to the liver and the root of the mesentery. A palliative gastrostomy was performed and the patient was well for 1½ years. A second exploratory operation, because of recurrence of symptoms and roentgen demonstration of pyloric obstruction, revealed an aortic aneurysm; the stomach and duodenum were normal. Most recently, Desiderio *et al.*[13] reported a patient with an aneurysm who developed nausea and vomiting resulting in hyperchloremic alkalosis and abdominal pain exacerbated by lying in the supine position. Extrinsic pressure on the transverse duodenum near the ligament of Treitz with dilatation of the proximal duodenum was demonstrated roentgenographically. At operation, a huge aortic aneurysm originating at the level of the renal arteries compressed the duodenum at the ligament of Treitz. Obstruction apparently is caused by fixation of the distal duodenum between the superior mesenteric artery and the enlarging aneurysm. One of our patients complained of epigastric postprandial pain which was relieved by vomiting, but the duodenum was not obstructed at surgery. Aneurysms should, however, be considered in the differential diagnosis of high intestinal obstruction in elderly patients.

In previous reports there has been no general agreement regarding the frequency of gastrointestinal symptoms with abdominal aortic aneurysms. Although some authors have found intestinal complaints to be common,[3,32,52] others[18,37,46,55] have recorded them infrequently. Sommerville *et al.*,[55] in a review of 172 patients with abdominal aneurysms, reported abdominal pain, weight loss, anorexia, nausea, vomiting, constipation, and diarrhea in only 19 per cent. In contrast, in our series 55 per cent of the patients had intestinal symptoms (Table 1). Abdominal pain was the commonest complaint and occurred in 24 per cent of the patients although preoperative aortic rupture had developed in only 2 cases. The type of pain was not specific. Most frequently, it was described as colicky or intermittent, and was located in the left lower quadrant or epigastrium. The paroxysmal nature of the pain has been known for more than 130 years.[5] The pathogenesis of such pain has, however, not been understood. It has been attributed to vertebral erosion,[52] and to pressure on paravertebral sympathetic nerves[5,7] and on the intestines.[37] Superior mesenteric artery insufficiency is probably not implicated because the typical postcibal pain pattern[17,38] was found in only 2 of our patients, and the aneurysm extended to the level of this artery in only 4 cases. However, inferior mesenteric artery insufficiency may be causative, for colicky left lower abdominal pain is the most frequent symptom of patients with left colon ischemia,[12] and was the pattern in several of our patients (Table 1).

Weight loss with abdominal aneurysms has been frequently recorded;[3,6,36,52,55] it occurred in 18 per cent of our patients, and was as much as 10 pounds in 1 month. It is probably related to pressure of the aneurysm on the intestines as it was a finding in 5 of the 7 patients in whom this was demonstrated. Constipation was a prominent symptom in Beatty's[5] often quoted case of abdominal aneurysm published in 1830. A rectal stricture had been suspected because of associated symptoms of abdominal distention and severe, colicky, left lower abdominal pain. Osler[48] subsequently observed that constipation was a common symptom of patients with abdominal aneurysms. Ten per cent of our patients developed constipation within a few months of the diagnosis of an aneurysm. Carcinoma of the colon had been suspected in 1 of them, a 58 year old man with a 2 month history of constipation, diminished stool caliber, and difficulty in defecation. At operation, a normal colon and inferior mesenteric artery occlusion were found. Pathologic confirmation of mesenteric arterial insufficiency in our other patients with constipation is not available.

The problem of vascular insufficiency of the left colon has recently been reviewed by Demos.[12] He found that one-fifth of the patients had intermittent constipation and diarrhea. Constipation in patients with inferior mesenteric artery occlusion and abdominal aneurysms has been recorded,[23,43] although diarrhea has been more common.[12,29,38] It would appear that these symptoms are related to left colon ischemia in patients with abdominal aneurysms. This hypothesis is made more tenable by the fact that the collateral circulation in these elderly patients is frequently compromised, and that the development of left colon infarction is more often insidious than elsewhere in the bowel.[43] Ischemia of the left colon followed by stricture formation after resection of an aneurysm was first described by Moore;[45] constipation in such a patient has also been reported.[44] Vascular insufficiency of the left colon developed in 9 per cent of 120 aneurysmectomies reported by Smith and Szilagyi,[54] but the incidence was approximately 1 per cent in most series.[12]

None of our patients were jaundiced, although in Case IV (Fig. 4*A*) displacement of the common duct was apparent. In 1891, Dickinson[15] reported the first case of compression of the common duct by an abdominal aneurysm producing jaundice; since then several cases have been recorded.[47]

An expansile abdominal mass was found in 59 per cent of our cases and a nonpulsatile mass in 7 per cent. Tenderness of the mass was present in only 2 patients; both had unruptured aneurysms. Enselberg,[18] in 18 of 29 cases of abdominal aneurysms, found that tenderness of the aneurysmal mass was not necessarily associated with rupture; although others[3,55] attribute tenderness to rupture. Persistent intense, abdominal pain[46] or severe backache, are, however, highly suggestive of aortic rupture,[3,6,55] or dissection.[31]

The earliest article on the roentgen diagnosis of abdominal aneurysm was by Böttner,[7] who described the use of pneumoperitoneum and gastrointestinal studies. Distention of the stomach and colon with air permitted visualization of the aneurysm. Morgagni had noticed that aneurysms could erode vertebral bodies while leaving the adjacent interspaces intact.[5] Scalloping of the left anterior aspect of the bodies of T11-L2, the roentgen counterpart of this phenomenon, has long been considered pathognomonic of abdominal aneurysms.[8,21,32,37] In 1927, Farmer[21] was the first to direct attention to the diagnostic importance of calcifications in the walls of aneurysms and also mentioned that they could erode the lower ribs. Klinefelter[35] emphasized the curvilinear character of the calcifications and their concave relation to the spine, and that they could be differentiated from aortic calcifications since the latter were frequently parallel. Lang[37] indicated that in contrast to arteriosclerotic, syphilitic aneurysms commonly become large without calcifying. Baer and Loewenberg[2] reported a patient with a large calcified thoracoabdominal aneurysm that had depressed the right lobe of the liver and had been mistaken for an hepatic echinococcus cyst. Elevation of the right lobe of the liver, and thus the right diaphragm, by an aneurysm has also been observed.[1] Grayson and Kennedy,[28] and Janower[33] have described the roentgen findings of ruptured abdominal aneurysms.

Curvilinear calcifications, nonparallelism of calcified aortic walls, and a paraspinal soft tissue mass were diagnostic of aneurysms in 58 per cent of the 57 preliminary abdominal roentgen examinations in our series. Lateral or left posterior oblique projections sometimes demonstrated the pathology when the frontal roentgenogram was normal or equivocal.

The psoas margin was distinct in all patients except the 2 in whom rupture of the aneurysm had occurred.

Giordano,[27] Galifi,[25] Unger and Poppel,[58] and less convincingly, Lins and de Morais[39] have published roentgenograms illustrating indentation of the lesser curvature of the stomach by aneurysms. Hubeny and Pollack[32] called attention to the value of gastrointestinal barium studies in patients with abdominal aneurysms, and published roentgenograms of a case showing extrinsic pressure on the gastric antrum, duodenum, and jejunum. Until the present study, the only other illustrations of gastrointestinal distortion by nonobstructing abdominal aneurysms were by Feldman[22] in 1945, Lang[37] in 1947, and Estes[19] in 1950.

In our series, 30 per cent (7 of 22 patients) had abnormal upper gastrointestinal studies. The roentgenograms of a patient examined recently are herein included because they illustrate the striking roentgen changes sometimes produced by abdominal aneurysms (Fig. 6, *A*, *B* and *C*). It is evident that only large aneurysms, right-sided apparently more than left, cause intestinal displacement. The gastrointestinal deformities caused by syphilitic and arteriosclerotic aneurysms can be contrasted because of the different levels at which these aneurysms arise. Those of syphilitic origin are usually found above the renal arteries[52] and impress the lesser curvature of the stomach. Arteriosclerotic aneursyms, however, usually begin several centimeters below the renal arteries; the most common roentgen appearance associated with them in our patients was elevation and pressure deformity of the third portion of the duodenum (Fig. 2*A*; 3*A*; 5*B*; and 6*A*). Extrinsic pressure on the greater curvature of the stomach (Fig. 2*A*; and 3*A*) and spreading of small bowel loops (Fig. 1*B*; 2*A*; and 6*A*) were also observed. In differential diagnosis pancreatic neoplasms or cysts, mesenteric cysts, abdominal lymph nodes enlarged by lymphoma, and retroperitoneal tumors must be considered. Differentiation from other retroperitoneal masses is possible because aneurysms are closely related to the spine and frequently contain calcifications. Whereas pancreatic neoplasms may widen the duodenal loop, aneurysms are more likely to narrow it by extending below and elevating the transverse duodenum (Fig. 2*A*; 3, *A* and *B*; 5, *A* and *B*; and 6, *A* and *B*).

SUMMARY AND CONCLUSIONS

Gastrointestinal symptoms with abdominal aortic aneurysms occur more frequently than previously supposed. In a retrospective analysis of 100 consecutive cases, diagnosed by intravenous aortography, such complaints were found in 55 per cent. The most common were abdominal pain in 24 per cent, weight loss in 18 per cent, and constipation in 10 per cent. Ischemia of the left colon is proposed as the cause of the constipation.

The diagnosis of aneurysm could be made in 58 per cent from the conventional abdominal roentgenograms. In the patients with gastrointestinal studies, roentgen abnormalities were found in 30 per cent. These were distortion, anterior and superior displacement of the third part of the duodenum, extrinsic pressure on the greater curvature of the stomach, and spreading of small bowel loops.

A review of the literature revealed only 8 cases of duodenal obstruction by aneurysms, usually near the ligament of Treitz. Jaundice with abdominal aneurysms is a rarity. Approximately 100 cases of rupture of aneurysms into the intestinal tract have been recorded, and in 60 per cent of these, it occurred into the transverse duodenum. In several cases early diagnosis and reparative surgery were curative.

Conventional abdominal roentgenograms are, therefore, essential in elderly patients with the mentioned gastrointestinal complaints, especially when associated with gastrointestinal hemorrhage. If these are unrevealing or equivocal, an upper gastrointestinal series may be helpful in establishing the diagnosis of aneurysm, which can be definitively made by aortography.

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ELECTROLYTE DEPLETION SYNDROME ASSOCIATED WITH VILLOUS ADENOMAS OF THE RECTOSIGMOID*

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VILLOUS adenomas of the colon present many interesting and unusual characteristics from a pathologic, clinical and roentgenologic point of view. It is important for the radiologist to be cognizant of the possible metabolic consequences which may be associated with this tumor. This has received scanty attention in the radiologic literature. They are benign, but have a great tendency to malignant degeneration, are usually large and bulky when discovered, and their distinctive gross anatomy leads to a roentgen appearance which is often typical, permitting a specific preoperative diagnosis. To this list of features there must be added a potential physiologic behavior of extreme clinical importance not associated with other neoplastic gastrointestinal lesions, that of extreme fluid and electrolyte depletion.

Frye[9] in 1959 reported a case in which electrolyte depletion was present, but little emphasis was placed on this aspect of the problem. Brief reference has recently been made to this syndrome by Barekman[1] and by Figiel and Figiel.[6] The condition was first reported by McKittrick and Wheelock[15] in 1954. Since then numerous cases have been reported in the surgical and medical literature.

REPORT OF A CASE

A 78 year old white female was admitted following a 2 week period marked by anorexia, nausea, vomiting, and subsequent development of vertigo, confusion and lethargy. It later was discovered that she had been having multiple loose bowel movements daily for several months.

Physical examination was unremarkable except for obvious lethargy, restlessness and diffuse muscular weakness. Rectal examination was deferred at the time of admission.

Laboratory studies of interest on admission included an elevated white blood cell count of 27,400, 2+ proteinuria, blood urea nitrogen of 219, sodium 106 mEq/L., potassium 3.0 mEq/L., chlorides 60 mEq/L., bicarbonate 6.1 mEq/L., and calcium 6.9 mg. per cent (see Table I).

Electrocardiograms showed St-T wave changes consistent with hypokalemia. Blood gas studies (pH 7.35, pCO_2 16.2 mm. of Hg) indicated a compensated metabolic acidosis. Azotemia was felt to be prerenal, with the acidosis and electrolyte deficiencies due to excessive loss of gastrointestinal secretions. Shortly after admission the patient became semicomatose and hypotensive requiring aramine for several days

Table I

LABORATORY DATA IN REPORTED CASE

Hospital Day	1	2	3	4	5	6	7	8	9	19	24
Blood urea nitrogen	219	194	194	112	110	68	45	29	19	10	8
Creatinine	11.2	7.2	5.8	—	3.3	2.1	—	—	—	—	—
Bicarbonate	6.1	16.6	18.4	20.4	19.5	21.3	23.3	21.5	17.9	31.5	26.5
Chloride	60	94	94	92	95	104	104	101	98	97	92
Sodium	106	122	135	134	131	136	140	136	128	138	128
Potassium	3.0	2.3	2.5	2.9	2.8	3.0	3.3	3.2	3.2	4.3	3.6

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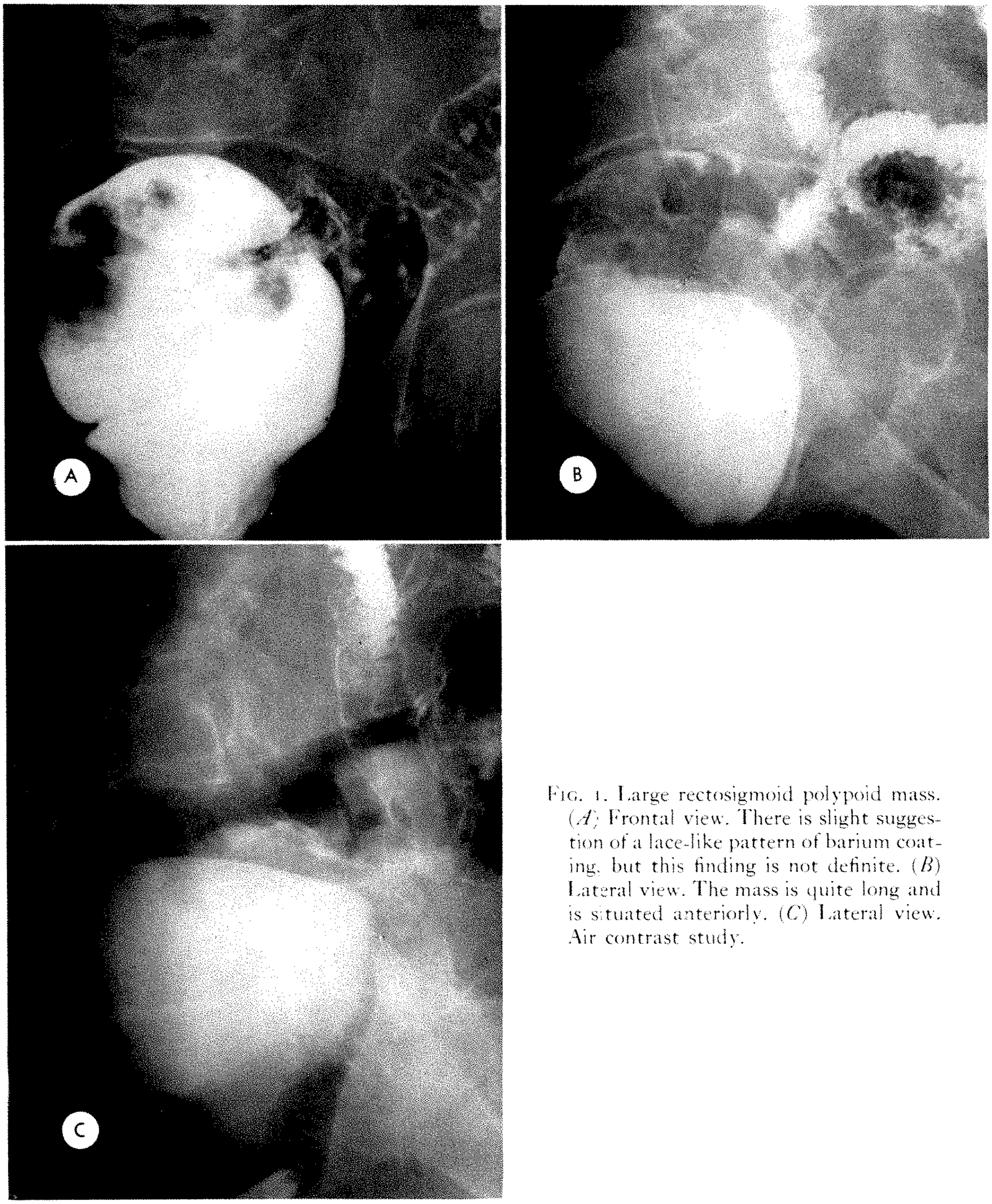

Fig. 1. Large rectosigmoid polypoid mass. (*A*) Frontal view. There is slight suggestion of a lace-like pattern of barium coating, but this finding is not definite. (*B*) Lateral view. The mass is quite long and is situated anteriorly. (*C*) Lateral view. Air contrast study.

to support blood pressure, and intensive fluid and electrolyte replacement was carried out. Watery diarrhea (800 to 1,500 cc. per day) persisted daily during all her hospitalization. A subsequent rectal examination revealed a large soft anterior rectal wall mass confirmed at proctoscopy at the 8 cm. level. Biopsy revealed a typical benign villous adenoma. Barium enema studies were then obtained, revealing a large polypoid mass in the upper rectum (Fig. 1, *A*, *B* and *C*).

An abdominal perineal resection was carried out, with an uneventful postoperative course, there being no further electrolyte problems. The surgical specimen revealed no areas of malignant change.

ROENTGEN FINDINGS

The typical roentgen appearance of villous adenoma is well known, consisting of a lace-like pattern produced by trapping of the barium in the interstices of the many frond-like projections of the tumor. The gross morphology of these tumors is not constant however, and the exuberance of the projections may not be sufficient to produce the typical pattern. These tumors are almost always quite bulky when discovered and the radiologist can suspect a villous adenoma when encountering an extremely large polypoid colonic mass. This situation was present in the patient reported here. The roentgenographic findings when combined with the syndrome of dehydration and systemic electrolyte loss are almost pathognomonic.

DISCUSSION

The clinical background associated with this syndrome is quite interesting. The patients often have a chronic watery diarrhea for long periods without other significant symptoms. They come to accept this almost as a "normal" way of life and do not get into difficulty as long as they can replace their fluid and electrolyte losses through adequate oral intake. When this replacement fails, such as after periods of increased activity, hot weather, or inadvertently prescribed low salt diets, the patients will rapidly sink into a severe depletion state presenting as a life threatening emergency. The history of the chronic diarrhea may not be immediately available to the attending physician. In addition, a rectal examination is often deferred in such a patient. In a number of the reported cases, the patients had sought medical help for the chronic diarrhea and no specific diagnosis had been made. Despite their size, these tumors are easily missed on rectal and proctoscopic examination due to their softness, pale color, and lack of resistance to the sigmoidoscope. All of these factors may delay recognition of the true nature of the problem. The metabolic picture in the acute depletion phase consists basically of dehydration, hyponatremia, hypokalemia, hypochloremia, prerenal azotemia, oliguria or anuria, metabolic acidosis, circulatory collapse, and in some patients hypocalcemia. All of these were present in the case reported here. The prerenal azotemia is related to the severe dehydration with its resultant decreased renal blood flow in addition to the increased catabolic state which these patients develop.

Shnitka and associates[18] reported an average duration of symptoms of 5.4 years prior to the emergency episode, with an average age at the time of discovery of 66 years. The mechanism of copious secretions is not definitely established, though it is, no doubt, at least partially related to the vast surface area these tumors possess. Microscopically, the lesion is composed of multiple villous projections covered by tall columnar mucus secreting epithelium. No qualitative or quantitative distinction has as yet been made between the secreting, and the more common nonsecreting tumors except that the functioning tumors have all been either rectal or rectosigmoid in location. This phenomenon suggests that some more proximal adenomas may be secreting, but that their discharges are reabsorbed in the colon distal to the lesion. Losses in the depletion syndrome represent 10 to 40 times the daily water, sodium, and potassium excreted in the stool of normal individuals. A villous adenoma may also occur as a coincidental lesion associated with other conditions producing chronic diarrhea such as ulcerative colitis. Figure 2, *A* and *B* shows the roentgenograms in such a patient, who, however, did not suffer from severe electrolyte loss.

TREATMENT

Treatment consists of vigorous fluid and electrolyte replacement and alkalinization with sodium bicarbonate. After the deficiencies have been corrected, adequate

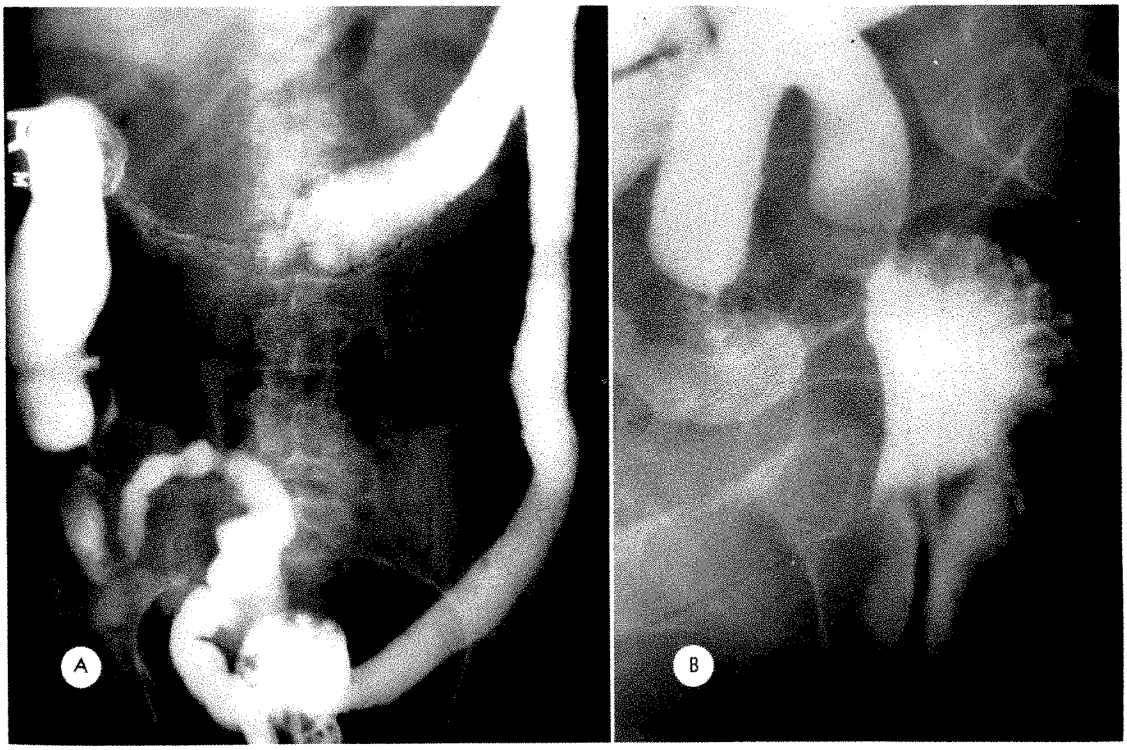

FIG. 2. Polypoid rectal mass with extensive ulcerative colitis. (*A*) Supine roentgenogram shows mass in rectum and extensive changes of ulcerative colitis. (*B*) Lateral roentgenogram of rectum demonstrates multiple projections of the villous adenoma with barium trapping in the intervillous spaces.

maintenance must be provided until the source of the losses is eliminated. Early diagnosis of the underlying lesion is essential and the radiologist must be aware of the important relationship between a rectal mass discovered on a barium enema study and the patient's metabolic difficulties. If the lesion remains undiscovered, the course will progressively worsen, and the cause for the systemic collapse will remain obscure, usually being confused with diabetic coma, adrenal insufficiency, or other metabolic emergencies. Without removal of the lesion, even the most vigorous replacement therapy may become ineffectual, leading to a fatal outcome. Temporarily, local hypothermia has been found effective in reducing secretions from the tumor.[18]

Ideally, correction of the depletion state should be attempted before surgery, but this may not always be possible and definitive therapy should not be unduly delayed. Total resection is the only effective remedy, diversionary colostomy is of no value, and fulguration, irradiation, and incomplete excision have all been followed by a high rate of recurrence.

In the postoperative state, another interesting electrolyte deficient condition may develop, of which the radiologist should be aware, that of hypokalemia. This deficiency produces severe intestinal distention, most marked in the large bowel, and is easily confused with intestinal obstruction.[13]

SUMMARY

1. Villous adenomas of the rectum or rectosigmoid occasionally secrete extremely large amounts of fluid and electrolytes leading to a severe depletion state.

2. There is usually a long history of chronic watery or mucous diarrhea, which may gradually, or more often rather suddenly, result in an acute syndrome characterized by circulatory collapse, dehydration, and severe deficiency of electrolytes.

The chronic diarrhea may not be immediately known in the emergency situation.

3. The roentgen features of these tumors are well known and fairly characteristic, consisting of either a lace-like pattern of barium in a mass, and/or an extremely large bulky mass.

4. Early recognition of the lesion and its relationship to the metabolic emergency is of great importance. Complete removal is essential and is curative provided no malignant degeneration with metastasis has occurred.

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THE INCLINED LATERAL DECUBITUS POSITION FOR CHOLECYSTOGRAPHY*

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THE lateral decubitus position for cholecystography was introduced and popularized by Kirklin.[1] This has proved to be an advance over the upright position which often resulted in intestinal contents obscuring the gallbladder. While recognizing this improvement, it must be admitted that all technical problems have not been resolved by the lateral decubitus view. Lubert and Krause[2] have advocated upright spot roentgenography with graded compression. Miller[3] has recently suggested that oblique studies in the lateral decubitus position are helpful. Our efforts to further refine cholecystography have led to the use of the inclined lateral decubitus position.

TECHNIQUE

The patient is asked to kneel on the table. A cotton pad, 9×9×17 inches in size, is placed to the right of the patient. With gentle assistance the patient is asked to lean to the right so that the right hip rests on the pad and the right shoulder on the table. The legs are supported by a second pad (Fig. 1). The resultant elevation of the hip is 7 to 8 inches. The roentgenographic exposure is identical to that used in the ordinary lateral decubitus position.

This view was used supplementing the three conventional views (posteroanterior, left anterior oblique and lateral decubitus) in 90 patients with opacified gallbladders. In 40 patients (44 per cent), the inclined lateral decubitus view provided no additional information over that observed on the regular lateral decubitus roentgenogram. In 48 patients (53 per cent), the inclined lateral decubitus study was distinctly better and was often essential in arriving at a firm diagnosis. In 3 examinations, the plain lateral decubitus roentgenogram was superior to the inclined decubitus roentgenogram.

DISCUSSION

The main advantage of the inclined lateral decubitus study was the separation of gas and contrast material within the bowel from the opacified gallbladder. It resulted in a much greater assurance that gallstones were not present (Fig. 2, *a* and *b*; 3, *a* and *b*; and 4, *a* and *b*). This additional view was also helpful in the diagnosis of gallstones. Of 15 patients with gallstones, there were 3 in which the certainty of the diagnosis rested on the inclined lateral decubitus study (Fig. 5, *a* and *b*).

There are two disadvantages to the inclined lateral decubitus position: it is somewhat awkward for the patient, and occasionally ribs or calcified costal cartilage become superimposed on the gallbladder.

The method has not been adopted for routine use; it is employed only when the regular lateral decubitus roentgenogram leaves the diagnosis in doubt.

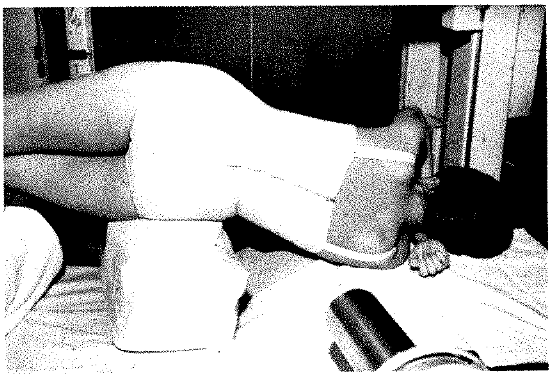

FIG. 1. Inclined lateral decubitus position. Hips elevated 8 inches. Legs supported.

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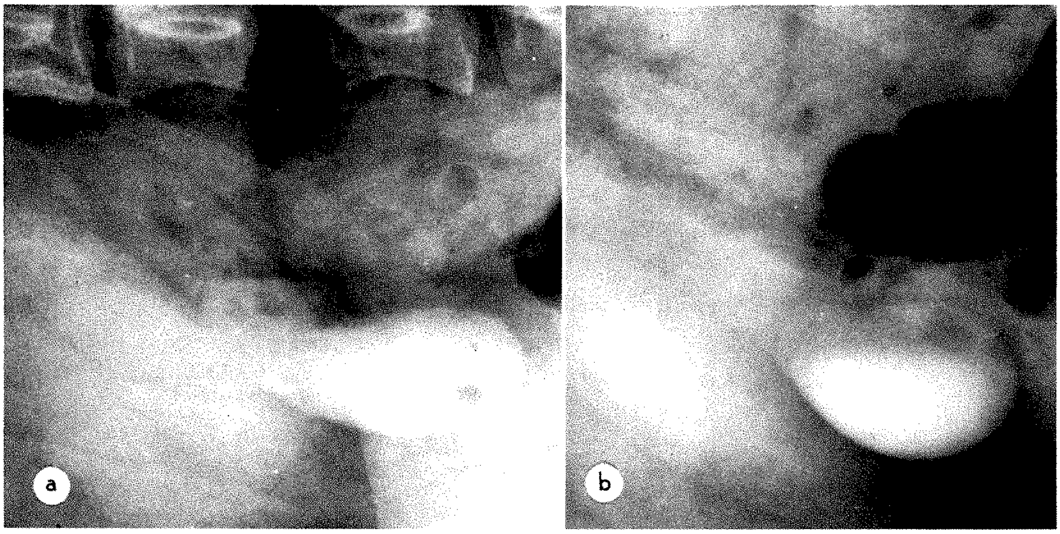

Fig. 2. (*a*) Regular lateral decubitus study. Gas obscures portion of gallbladder. (*b*) Inclined lateral decubitus study. Improved appearance of gallbladder.

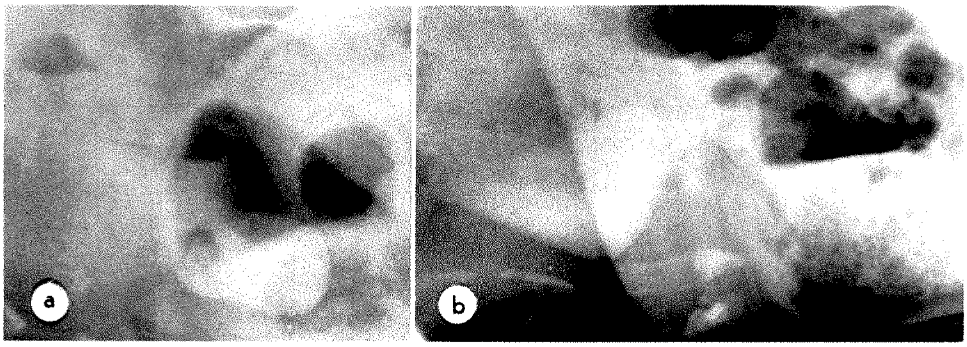

Fig. 3. (*a*) Regular lateral decubitus study. Gas and fecal material superimposed on gallbladder. (*b*) Inclined lateral decubitus study. Gallbladder moves more cephalad, away from the hepatic flexure of the colon.

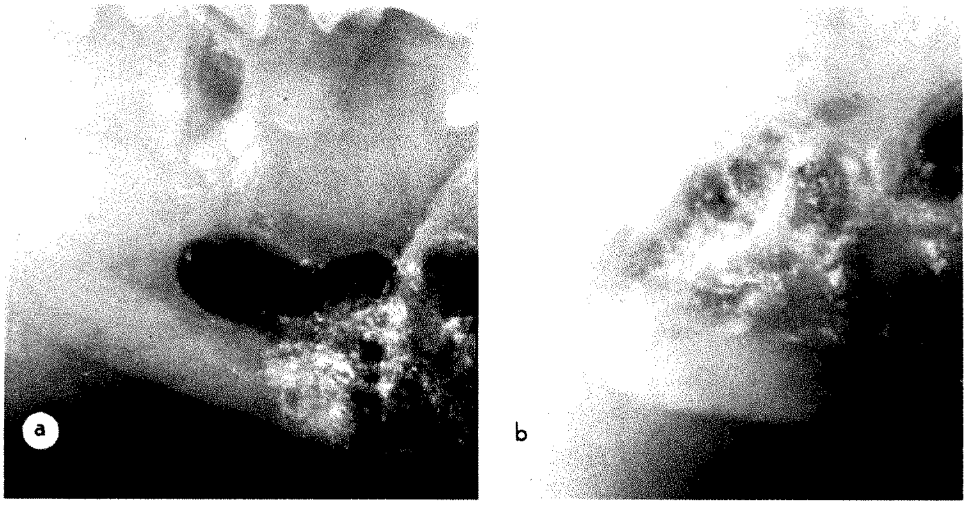

Fig. 4. (*a*) Regular lateral decubitus study. Contrast material obscures fundus of gallbladder. (*b*) Inclined lateral decubitus study. Good visualization of gallbladder.

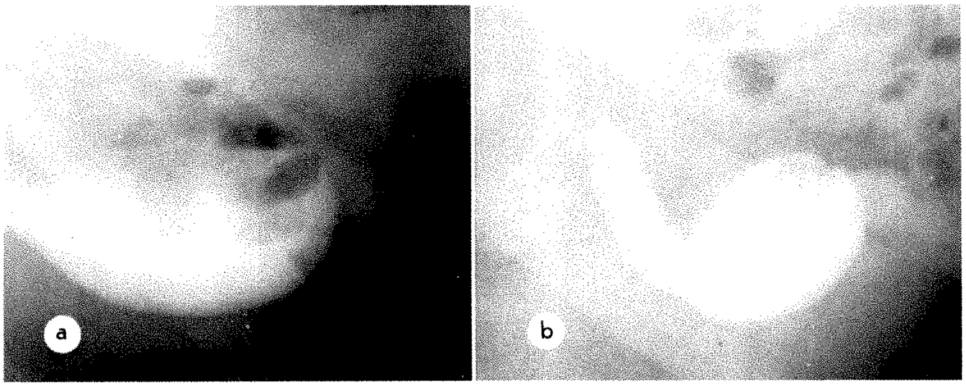

Fig. 5. (*a*) Regular lateral decubitus study. Gas over gallbladder (stones?). (*b*) Inclined lateral ducubitus study. Gas away from gallbladder. Two large floating stones are seen. Confirmed by cholecystectomy.

CONCLUSION

A posteroanterior exposure of the opacified gallbladder made in the lateral decubitus position with the hips elevated 7 to 8 inches has been found helpful in complementing the conventional cholecystographic examination.

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PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY

A PRELIMINARY REPORT DESCRIBING A NEW APPROACH TO THE EXAMINATION

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PERCUTANEOUS transhepatic cholangiography is not a new procedure. The first transabdominal puncture was performed in 1921, by Burckhardt and Müller;[1] in their case, the needle was inserted into the gallbladder and a cholangiogram was obtained in this fashion. Huard and Do-Xuan-Hop,[4] in 1937, were the first to report a technique of percutaneous transhepatic cholangiography; however, their paper does not clearly define the landmarks and the exact procedure which was used. The first description of percutaneous transhepatic cholangiography in the western literature appeared in 1952 by Carter and Saypol.[2] Their cutaneous landmark was the subcostal area in the mid-clavicular line. In the past few years much has been written about this examination, especially by Glenn *et al.*[3]

ANATOMY

The divisions of the biliary tree are relatively simple. The right hepatic duct is divided into a dorsocaudal and a ventrocranial branch. At the most superior aspect of the dorsocaudal branch, there is a secondary take-off cephalad which is called the ascending ramus. The left duct has no divisional nomenclature (Fig. 1). Figure 2 is a T-tube cholangiogram showing the more anterior position of the left ductal system in relationship to the more posteriorly located right ductal system. In addition, the left ductal take-off has a more acute angulation. It has long been felt that the acute angulation as well as the longer extrahepatic course results in earlier and greater dilatation of the left hepatic duct in extrahepatic bile duct obstruction.[5] The junction of the cystic and common hepatic ducts forms the common bile duct which passes behind the distal duodenal bulb into the pancreatic region. The duodenal intramural portion is called the ampullary segment.

TECHNIQUE

The technique employed consists of 3 parts: (1) puncture of the left intrahepatic biliary tree; (2) attempt to determine the level of obstruction, by dividing the common bile duct into prepancreatic, pancreatic and ampullary. This is done by the use of a mercury filled Miller-Abbot tube; and (3) performance of correlative gastrografin duodenal sweep studies when indicated.

Because of the closer proximity of the left ductal system to the anterior abdominal wall as compared to the more posteriorly located right ductal system, it seemed to us that this would be the more favorable site for percutaneous puncture, especially since the left duct is the first to dilate and most often attains the greater size. Carter and Saypol[2] inserted the needle into the left lobar ductal system, but the landmarks are not clearly defined. The mid-clavicular line just below the right costal margin with 45° angulation toward the skin and directed medially was chosen by Glenn *et al.*[3] Another technique using cutaneous landmarks is that described by Burckhardt and Müller.[1]

It is felt by the present authors that a

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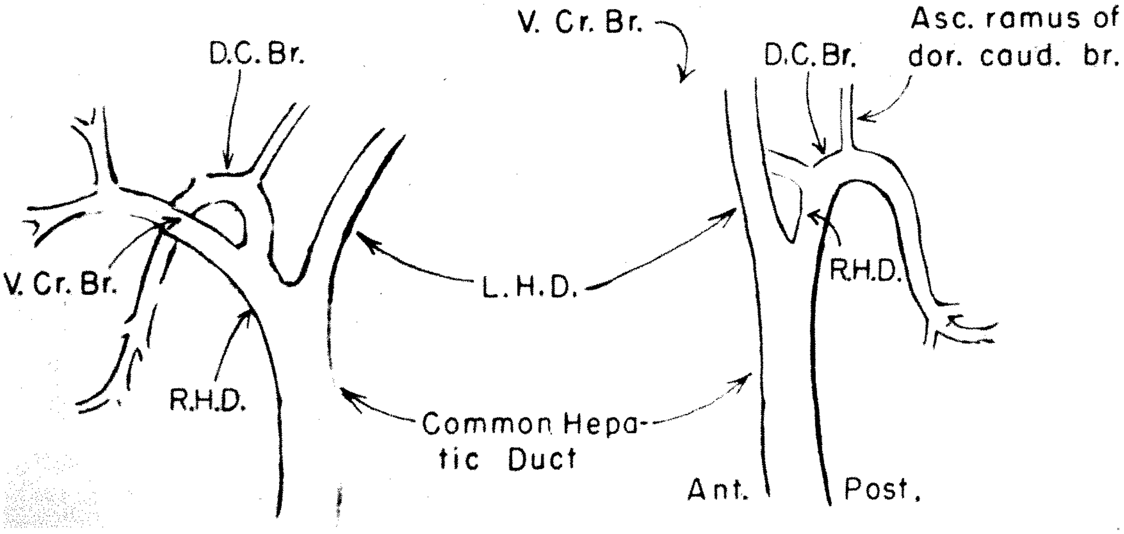

FIG. 1. Diagram showing biliary tree in the frontal and lateral projections. V. Cr. Br. = ventrocranial branch; D. C. Br. = dorsocaudal branch; L. H. D. = left hepatic duct; and R. H. D. = right hepatic duct. (Courtesy of Dr. W. Z. Stern and co-workers.[5])

correlation of the cutaneous landmarks with the right or left ductal system puncture would have a definite advantage and that association with combined correlative studies by means of gastrointestinal opaque media would further enhance the accuracy of the examination. Accordingly, the following technique was devised by one of the authors (J. Joison) and introduced as a clinical procedure in performing percutaneous transhepatic cholangiography.

The patient is prepared for the examination with the insertion of a Miller-Abbot tube which is advanced so that the tip is beyond the ligament of Treitz. The lumen of the tube is filled with mercury for fluoroscopic and roentgenographic control. When the tube is in place, it encircles the head of the pancreas. With the aid of fluoroscopy, the course of the tube is marked on the skin of the anterior abdominal wall. We have found that television monitoring has a distinct advantage over the use of routine fluoroscopy, because of ease of operation and the illumination of the room during the examination. The points of junction of the first and second portions, as well as the second and third portions of the duodenum, are marked on the skin and connected by a straight line, which is called the superior-inferior duodenal cutaneous line. This cor-

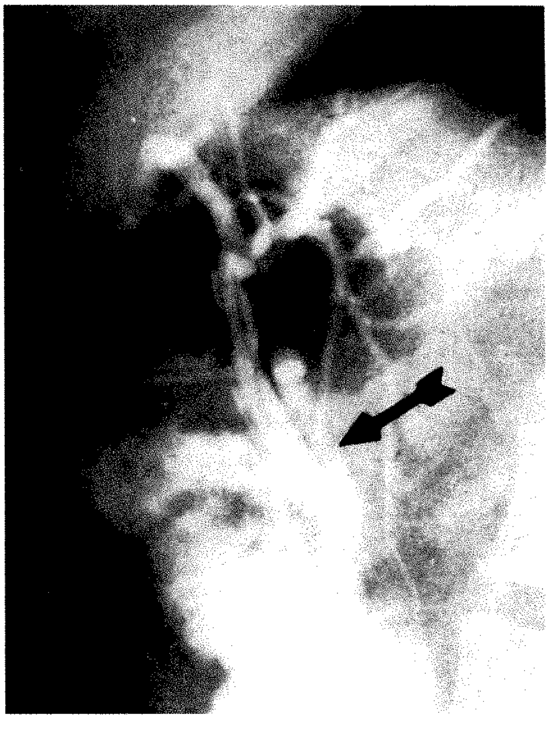

FIG. 2. T-tube lateral cholangiogram showing anterior position of the left ductal system. The arrow indicates a posterior long cystic duct stump. (Courtesy of Dr. W. Z. Stern and co-workers.[5])

responds very roughly to the second portion of the duodenum, but, of course, is not convex laterally. The percutaneous puncture is made with a No. 17 spinal needle 2 finger breadths directly above and parallel to the vertical superior-inferior duodenal cutaneous line with a 45° cephalad angulation to the skin (Fig. 3). Using this approach we have consistently entered the left intrahepatic biliary tree. With the mercury filled Miller-Abbot tube encircling the head of the pancreas, the level of obstruction is defined as prepancreatic, pancreatic (this, of course, falls within the confines of the concave loop of the Miller-Abbot tube), and ampullary (when the obstruction is in the intramural portion or ampullary segment of the common bile duct). It is also possible with the Miller-Abbot tube in position to withdraw the distal portion of the tube either to the duodenal cap or to the stomach, and obtain studies with gastrografin or other contrast media for correlation of the duodenal sweep.

MATERIAL

Table I gives the lesions studied in our series of patients. In 9 of the 13 patients, the technique described above was used. Of the 13 patients, 11 were explored either on the same day or the following morning, and 2 were considered inoperable. One of the 2 inoperable patients died of biliary peritonitis. The second was not explored because he was thought to have inoperable carcinoma; he had no sequelae after the cholangiography.

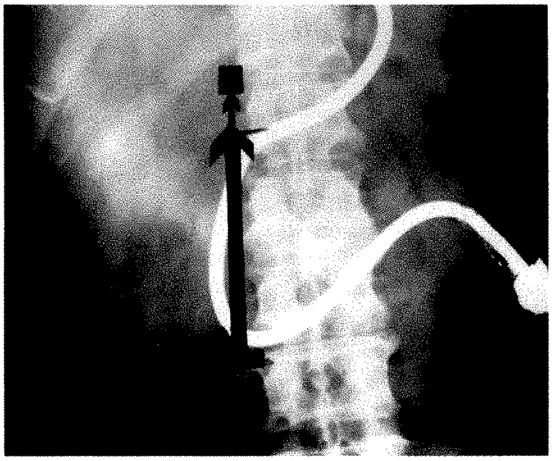

Fig. 3. Roentgenogram showing the Miller-Abbot tube encircling the head of the pancreas; the superior-inferior duodenal cutaneous line (large arrow); and the puncture site (small arrow).

Table I

SUMMARY OF CASES

Roentgen Findings	No. of Cases
Pancreatic carcinoma	6
Hepatic duct carcinoma	1
Common bile duct carcinoma	2
Ampullary carcinoma	1
Stones, common bile duct	1
No proof	2
Total	13

The following 4 cases illustrate the application of our technique.

REPORT OF CASES

Case 1. Q.S., B.C.H. #38143. A 72 year old Chinese male entered the Boston City Hospital with a history of persistent jaundice of 2 months' duration. Other symptoms were anorexia, pruritus, postprandial fullness below the xiphoid, and a 15 lb. weight loss. He denied fever, chills and abdominal pain. Physical examination revealed deep icterus of the sclerae and skin. The liver was 1 finger breadth below the right costal margin. Stools were guaiac positive. Laboratory studies showed bilirubin values from 18.8 to 27.5 mg. per cent and alkaline phosphatase of 6.8 Bodansky units.

Transhepatic percutaneous cholangiography was done prior to exploration. Figure 4 shows the needle positioned in the tertiary division of the left lobar duct. Figure 5 demonstrates marked dilatation of the intrahepatic biliary radicles and a greatly dilated common bile duct down to its distal portion. Without air or other contrast media in the duodenal sweep, the exact level of obstruction was difficult to delineate. In Figure 6, the Miller-Abbot tube is in position and the area of obstruction of the distal common bile duct is seen in the region of the ampulla of Vater. The diagnosis of cancer of the ampulla was considered most likely and

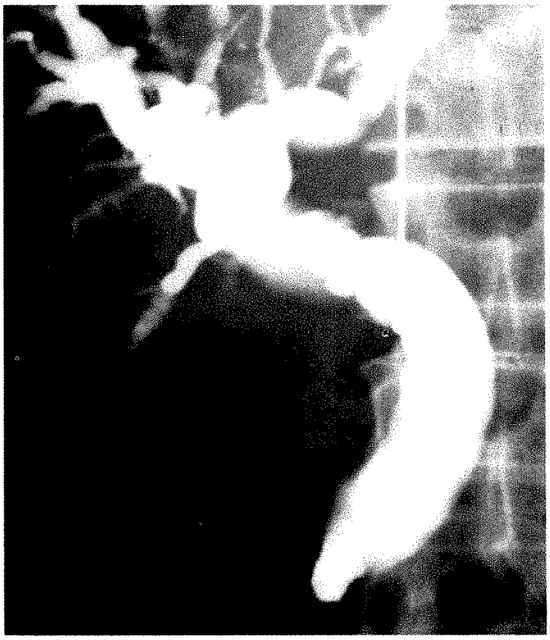

Fig. 4. Case i. Carcinoma of the ampulla of Vater. Percutaneous transhepatic cholangiogram showing the needle in the left tertiary division.

the patient was explored. Pathologic studies showed a 1.8 cm. mass at the ampulla which, on microscopic examination, proved to be a carcinoma of the ampulla of Vater.

Case ii. J.H., B.C.H. #42809. A 62 year old female entered the Boston City Hospital with a history of 5 to 6 weeks of jaundice with ill-defined back pain. Physical examination revealed hepatomegaly; the liver was palpated 2 finger breadths below the right costal margin. The patient was markedly icteric. Laboratory studies showed an alkaline phosphatase of 6.8 to 9.8 Bodansky units; bilirubin values ranged from 20 to 25 mg. per cent.

Using the described technique, transhepatic percutaneous cholangiography was done. Figure 7 shows the needle in the left ductal system. In Figure 8, with the Miller-Abbot tube encircling the head of the pancreas, the level of obstruction is seen to be in the region of the head of the pancreas. Figure 9, a combined gastrografin study, shows clearly the enlargement of the head of the pancreas with pressure along the greater curvature of the distal antrum and prepyloric region of the stomach as well as a slight irregularity along the lesser curvature. There is also some marked narrowing of the supra-ampullary portion of the second part of the duodenum. The common bile duct obstruc-

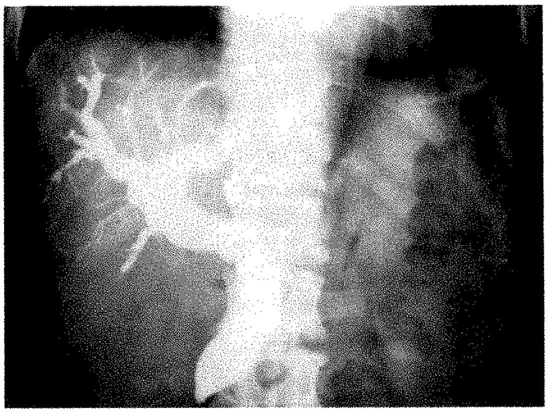

Fig. 5. Case i. Carcinoma of the ampulla of Vater. Percutaneous transhepatic cholangiogram showing the marked intrahepatic duct dilatation. The level of obstruction may be pancreatic or ampullary.

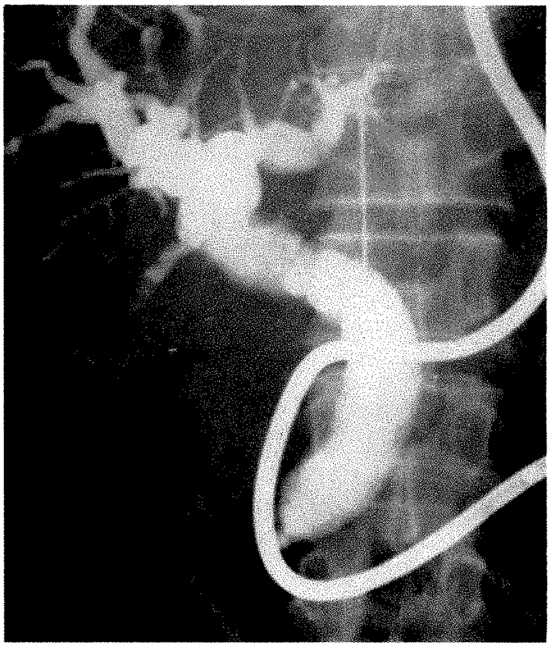

Fig. 6. Case i. Carcinoma of the ampulla of Vater. Percutaneous transhepatic cholangiogram showing the Miller-Abbot tube in position. There is marked dilatation of the ductal system with complete obstruction of the distal common duct. The tip of the obstruction is seen to terminate at the ampullary level as correlated with the tube in the duodenal loop.

tion is well demonstrated. The diagnosis of malignancy of the pancreas was considered most likely. The patient was explored and cancer of the pancreas which had actually invaded the greater curvature of the stomach was found.

Case III. A.M., B.C.H. #45496. A 52 year old female entered the Boston City Hospital with a history of slowly progressive jaundice over a period of about 3 to 4 weeks. Otherwise the patient felt well. Physical examination revealed marked icterus with no other abnormal findings. The liver was felt to be normal in size. Laboratory studies showed an alkaline phosphatase from 6.8 to 8.8 Bodansky units; bilirubin values ranged from 16 to 24 mg. per cent.

Transhepatic percutaneous cholangiography was done with the needle being inserted into the left lobar duct. In this particular case, a catheter was passed through the needle (Fig. 10). The level of obstruction, determined by using gastrografin which outlined the duodenal sweep, was found to be prepancreatic in type, located approximately 1 to 2 cm. above the region of the pancreas as shown in Figure 11. In view of the combined correlative study, the possibility of a bile duct carcinoma was considered. The level of obstruction was felt to be too high to be explained on the basis of ex-

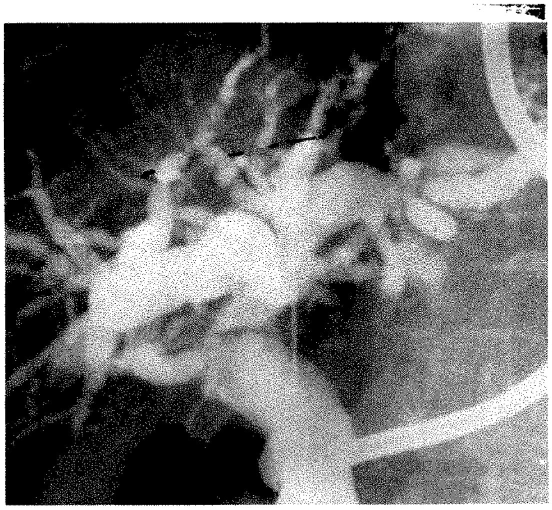

Fig. 7. Case II. Carcinoma of the pancreas. Percutaneous transhepatic cholangiogram showing the needle in the left ductal system. The Miller-Abbot tube is still in position. There is marked dilatation of the intrahepatic biliary radicles as well as extrahepatic duct dilatation.

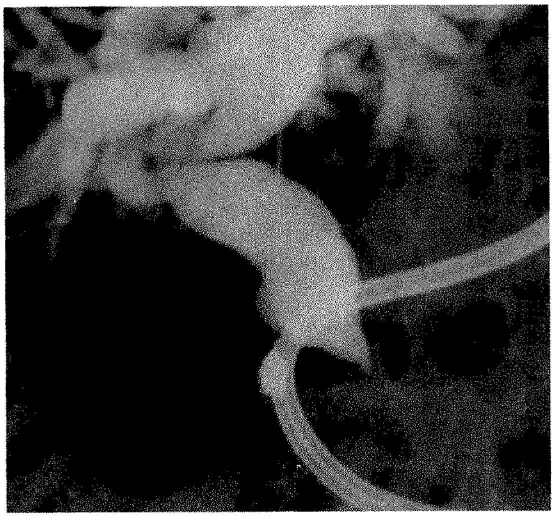

Fig. 8. Case II. Carcinoma of the pancreas. Percutaneous transhepatic cholangiogram. With the Miller-Abbot tube in position, the site of obstruction is shown to be at the pancreatic level.

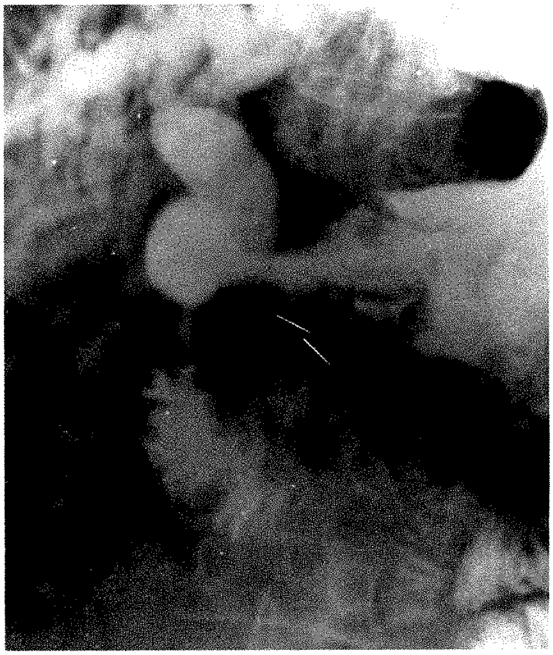

Fig. 9. Case II. Carcinoma of the pancreas. Percutaneous transhepatic cholangiogram with combined gastrografin study. The level of obstruction (arrow) is pancreatic. The pad effect of the stomach, the associated marked narrowing of the supra-ampullary portion of the duodenum and the obstructed common bile duct are consistent with a pancreatic neoplasm.

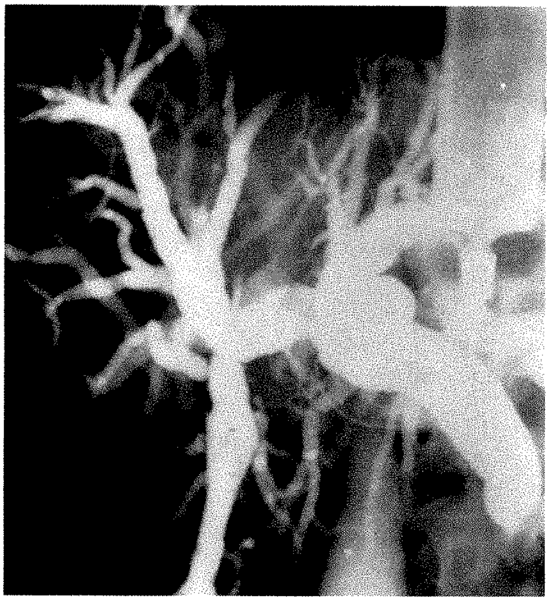

FIG. 10. Case III. Adenocarcinoma of the common bile duct. The catheter is in the left biliary intrahepatic tree. Marked intra- and extrahepatic duct dilatation is seen. The level of obstruction is questionable, but appears to be prepancreatic (see Fig. 11).

trinsic pancreatic invasion of the bile duct. The patient was explored and found to have an adenocarcinoma of the common bile duct.

CASE IV. N.L., B.C.H. #48197. A 48 year old female entered the Boston City Hospital with a history of jaundice of unknown duration. She had complained of frequent back pains during the previous 2 to 3 years. Physical examination revealed hepatomegaly; the liver was palpated 3 finger breadths below the right costal margin. The patient was markedly icteric. Laboratory studies showed an alkaline phosphatase of 7.2 to 8.9 Bodansky units; bilirubin values ranged from 18 to 28 mg. per cent.

Transhepatic percutaneous cholangiography was done and the needle was inserted into the left ductal system. Marked dilatation of the intrahepatic biliary radicles was found. The level of obstruction was demonstrated to be quite high, in the region of either the common hepatic duct or the proximal common bile duct (Fig. 12). In view of this, it was felt that the Miller-Abbot tube localization and the gastrografin correlative study were not indicated. The patient was explored and multiple gallstones in the common bile duct were found. Figure 13 shows the multiple areas of radiolucency with interspersed opaque medium in

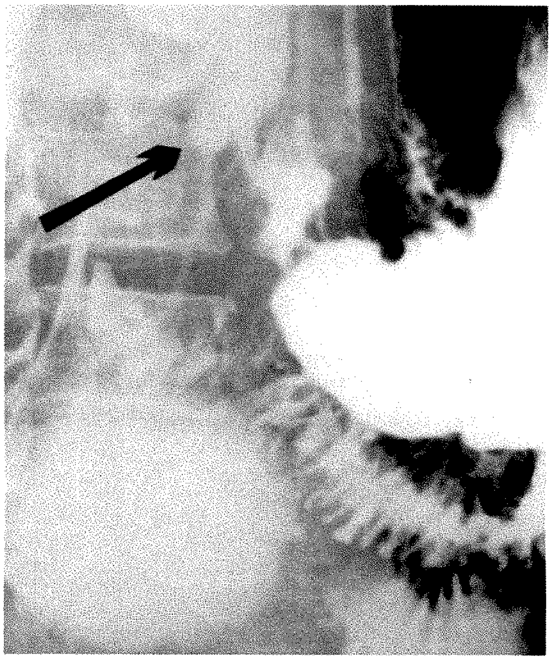

FIG. 11. Case III. Adenocarcinoma of the common bile duct. Percutaneous transhepatic cholangiogram showing prepancreatic level of obstruction. The duodenal loop is outlined with contrast medium. This confirms that the level of obstruction (arrow) is above the intrapancreatic portion of the duct as suspected in Figure 10.

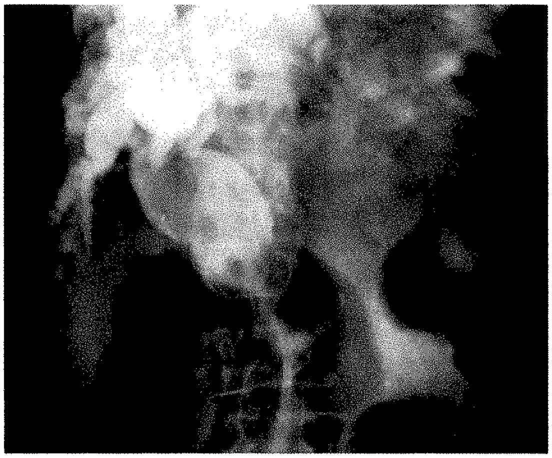

FIG. 12. Case IV. Multiple stones in the common bile duct. Percutaneous transhepatic cholangiogram showing marked intrahepatic biliary dilatation with complete obstruction in the common duct.

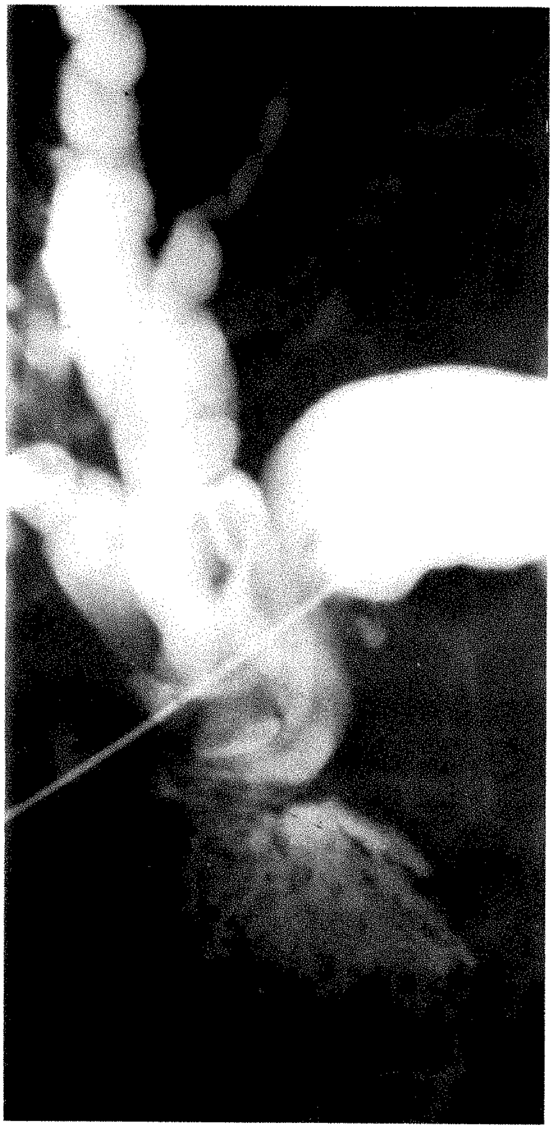

Fig. 13. Case iv. Multiple stones in the common bile duct. Percutaneous transhepatic cholangiogram showing multiple radiolucent defects (sludge) in the biliary tree at the origin of the common duct (prepancreatic obstruction).

the proximal portion of the duct, which represents sludge.

DISCUSSION

From our experience, we feel that the left ductal system can be selectively punctured. The early and greater dilatation and the more anterior postion of the left ductal system in relationship to the more posteriorly located right ductal system make this the preferred site. Percutaneous transhepatic cholangiography, with simultaneous use of a mercury filled Miller-Abbot tube, can aid in determining the level of obstruction which may be prepancreatic, pancreatic, or ampullary. Further information can be obtained by selective studies of the duodenal sweep with gastrografin or other contrast media. Such information may be of considerable value to the surgeon. We have found that the slightly longer time required for the procedure has not produced any significant increase in untoward reactions in the 13 patients in our series.

Two other points of interest might be mentioned here. One is the use of the catheter for biliary drainage. Percutaneous transhepatic cholangiography can be utilized not only for diagnosis, but may also serve as a decompressive procedure prior to surgery. It is felt that in the future a catheter rather than needle insertion will be the method of choice. Secondly, the possibility of further therapeutic application of percutaneous transhepatic cholangiography may conceivably be in the dissolution of stones in the common or intrahepatic bilary ducts.

SUMMARY

A preliminary report is presented describing a new technique for performing percutaneous transhepatic cholangiography.

By employing the described cutaneous landmark, which is obtained by the use of a mercury filled Miller-Abbot tube, the left ductal system can be punctured in its intrahepatic portion.

The levels of obstruction of the common bile duct have been defined as prepancreatic, pancreatic and ampullary.

Correlative gastrografin gastrointestinal studies with particular reference to the duodenal sweep are also of definite value.

It is hoped that further study along the lines described will bear out the desirability of using this method in the examinations of patients with obstructive jaundice.

ADDENDUM

Since completion of this report, 7 more cases have been done using the described technique with good results (Total 20 cases).

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CANINE CINEPORTOGRAPHY*

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THE hepatic circulation, in its complexity, inaccessability and interrelation with splanchnic and systemic hemodynamics, has been the object of many investigative approaches for several decades. Roentgenographic contributions to the study of the hepatic circulation were initially limited to static demonstrations of gross hepatic vascular morphology by means of opaque substances injected into anatomic specimens. With the development in the last 15 years of the serial film-changing devices and cinefluorography, it has become possible to study the hepatic nonarterial circulation dynamically. As a result, clinical utilization of such valuable roentgenographic procedures as splenoportography and percutaneous and retrograde hepatic venography has been stimulated.

Application of modern roentgenographic techniques to basic investigation of hepatic circulatory physiology is perhaps best exemplified by the work of Daniel and his colleagues.[5,6,7] Using small animals with normal and cirrhotic livers, these investigators were able to demonstrate the effects of various stimuli on transhepatic circulation time, on vessel caliber, on number of branch vessels opacified and on sinusoidal perfusion (*i.e.*, restricted intrahepatic circulation). Tornvall and Nordenström[10] used photofluorodensitometry to measure the cardiohepatic circulation time and the duration of sinusoidal perfusion. In 1960 blood flow in the hepatic veins of small animals was first visualized cinefluorographically by Brauer *et al.*[3] following transhepatic administration of radiopaque materials. No other reports dealing with similar or related uses of cinefluorography have been found by the authors.

In view of the above, it was decided to investigate the applicability of cinefluorography to the study of the hepatic nonarterial circulation in large animals and, if possible, to record cinefluorographically circulatory changes resulting from various pharmacologic stimuli. The dog was selected since its hepatic circulation has been extensively studied. In order to avoid direct trauma to the liver and to ensure uniform antegrade venous perfusion, the established technique of portal venous catheterization was employed.

METHODS AND MATERIALS

1. *Animals.* Seventeen adult mongrel dogs weighing 12–25 kg. were used. Each had been maintained on a standard large animal diet for at least several weeks before investigation. Four of these animals had each received a total of 32 intramuscular injections of carbon tetrachloride (1.0 ml.) during a 16 week period ending 1 year before investigation. Sixteen animals were in the fasting state when examined.

2. *Surgical Technique.* Following induction of anesthesia by intravenous pentobarbital (35 mg./kg.), the peritoneum was opened through a midline abdominal incision. The liver was inspected and palpated. A suitable vein in the jejunal mesentery was freed and ligated with fine silk. A second ligature was loosely tied 2 cm. cephalad to the first. A beveled No. 190 polyethylene catheter with end hole and preterminal side holes was then inserted into the peritoneal cavity through a left flank stab wound. The catheter was introduced into

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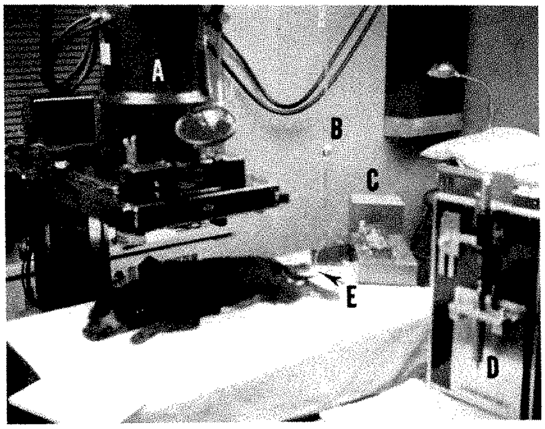

Fig. 1. Typical arrangement for cineportography: the anesthetized animal lies beneath the image amplifier with attached cine (A); the indwelling portal venous catheter (E) emerges from a flank stab wound and is attached to the saline manometer (B). At hand are the constant-speed infusion pump (C) and the automatic hydraulic syringe (D).

the mesenteric vein through a small incision between the ligatures and was advanced until its tip lay within the extrahepatic portion of the portal vein. The second ligature was then tightened so as to secure the catheter firmly within the vein without compressing the catheter. Patency of the catheter was maintained by filling with a heparin-saline solution from an attached reservoir syringe. The abdomen was closed with a running suture. The operative procedure usually required not more than 30 minutes. The level of anesthesia attained during surgery was maintained throughout the subsequent cinefluorographic studies.

3. *Cinefluorography.* The anesthetized catheterized animal was placed on the cinefluoroscopic table in prone position maintained by axillary pads (Fig. 1). The free distal end of the portal venous catheter was attached to an automatic hydraulic injector calibrated to deliver 20 ml. of 50 per cent hypaque in 2 seconds at room temperature. The volume of hypaque delivered to the portal vein with each injection was 1 ml. per kg. of body weight plus 3 ml. for catheter dead space.

Each injection of contrast material was monitored through an 8 inch image intensifier and was simultaneously recorded on Cine-Ray film exposed at the rate of 30 frames per second. Cinefluorographic exposure factors were kept constant for each series of injections in a given animal. The exposed cine for each animal was developed in an automatic film processor. Cinefluorographic studies were usually completed within 2 hours after laparotomy.

4. *Pharmacologic Agents.* The drugs administered, their doses, the intervals between administrations and the intervals between injection and subsequent cinefluorography are shown in Table I. Acetylcholine, epinephrine, norepinephrine and

Table I

Agent	Dose	Method of Administration	Pharmacologic Effect	
			Peak	Duration
Acetylcholine	10 μg./kg.	rapid intravenous	1 min.	10 min.
Epinephrine	5 μg./kg.	rapid intravenous	1 min.	10 min.
Norepinephrine	5 μg./kg.	rapid intravenous	0.5 min.	10 min.
Histamine base	30 μg./kg.	20 min. infusion	—	15 min.
Pitressin, aq.	0.5 unit/kg.	rapid intravenous	1.5 min.	20 min.
Sulfobromophthalein	Loading: 75 mg./kg. Infusion: 0.75 mg./kg.	rapid intravenous 20 min. infusion	—	—
Casein hydrolysate (5% and 20%)	Loading: 7.5 gm. Infusion: 5–10 gm.	rapid intravenous 30 min. infusion	—	—

All agents were administered via the portal vein. Cine sequences were begun at the time of predicted peak pharmacologic effect, or on completion of infusion. The sulfobromophthalein and casein hydrolysate infusions were continued at the same dose rate after baseline postinfusion cineportography, interrupted by administration of active agents and subsequent cineportography. Double doses of drugs were given to 3 animals.

pitressin were injected rapidly into the portal catheter through a 3 way stopcock followed by a flushing bolus of isotonic saline. For administration of histamine base, sulfobromophthalein and casein hydrolysate,* a motor-driven constant-infusion pump was employed. "Priming" doses of sulfobromophthalein or casein hydrolysate were rapidly administered immediately prior to the constant infusion of these agents. Not all agents were administered to each animal.

5. *Portal Venous Pressure.* Changes in relative portal venous pressure were measured with a saline manometer to which the portal catheter was attached intermittently. The level of the manometer was constant during investigation of each animal; no attempt was made to find the zero point.

6. *Pathologic Studies.* Open liver biopsies were obtained in 14 animals at the end of the experimental procedure. Hepatic tissue was immersed in 10 per cent formalin solution and was examined histologically after staining with acid fuchsin and hematoxylin-eosin.

7. *Cinefluorographic Analysis.* The film sequences of each animal were subjectively analyzed by multiple repeated viewings, employing variable projection speed, single-frame advancement, reversal of film direction, etc., as necessary.

Portal clearance times were measured by stop-watch timing, and were reproducible within 0.5 seconds. Multiple measurements were averaged for a final value. The portal clearance time was the time required for an injected bolus of contrast material to pass the portal bifurcation (or trifurcation). Correction was made for differences between cine exposure and projection speeds.

In efforts to quantitate regional sinusoidal perfusion, a target densitometer was placed behind and in contact with a translucent plastic screen upon which the cine sequences were projected. The line voltage supply to the recording densitometer was passed through a stabilizer. Densitometric readings were continuously and automatically recorded on rectilinear chart paper moving uniformly at 12 inches per minute. The system was calibrated before each use. The target densitometer position, cine projection speed, and film-screen distance were kept constant. Correction was made for the difference between film exposure and film projection rates. However, these densitometric measurements were not accurately reproducible. The apparatus did not permit direct photodensitometric measurements of loci on individual film frames.

It was not possible to measure accurately the variations in caliber of larger vessels either on the film or on the projected image.

RESULTS

Of the 14 animals whose livers were examined histologically, 10 were normal and 4 were cirrhotic. The microscopic changes of cirrhosis were slight or minimal in 3 animals and moderate in one. Ascites was not observed. The livers of the 3 animals not examined histologically were grossly normal.

Two types of information were obtained from this investigation: qualitative or subjective impressions derived from detailed and integrated viewing of the cine record and quantitative data based on measurements described above.

A. *Qualitative Analysis* (Fig. 2–7). Cineportograms clearly and graphically demonstrated the first 2 stages of antegrade hepatic venous blood flow, *i.e.*, flow of the radiopaque agent into the portal vein and portal branches, and subsequent sinusoidal perfusion. The hepatic venous efflux was not regularly nor well demonstrated cinefluorographically; the extent of opacification of the hepatic veins and inferior vena cava appeared to depend to a large extent upon the pharmacologic agent that preceded the film sequence. This result was in contrast to the regular demonstration of efferent hepatic blood flow by serial roentgenograms (Fig. 8, *A* and *B*). Variations in portal venous caliber were perceptible. In

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* Supplied by Baxter Laboratories, Inc., Morton Grove, Ill.

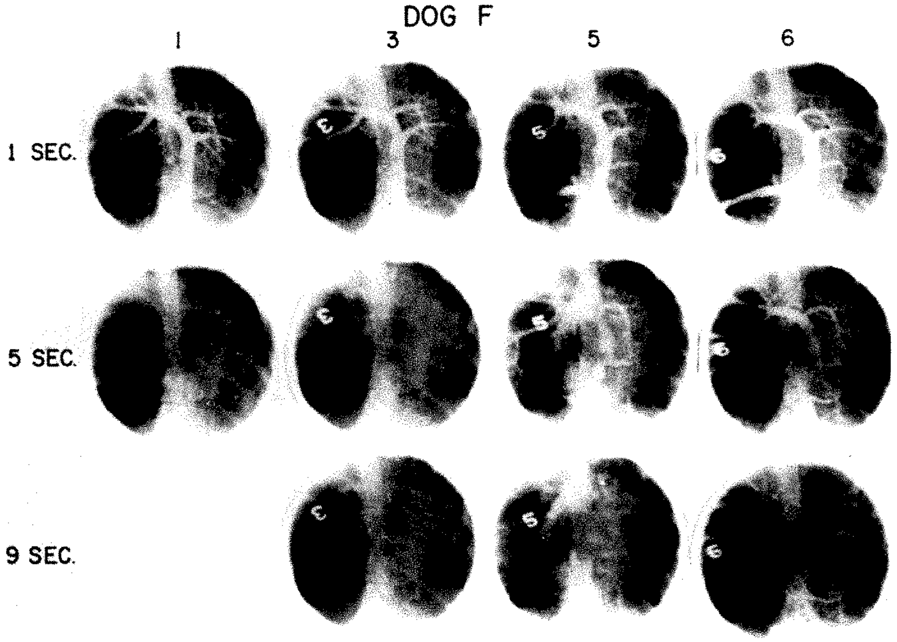

FIG. 2. (Dog F; normal animal.) Individual cine frames exposed exactly 1, 5 and 9 seconds after intraportal injection of contrast material. Cine excerpts are displayed vertically with numerical designations as follows: Run #1: baseline study; run #3: following epinephrine; run #5: following histamine; run #6 following pitressin. Double doses of drugs were employed (see Table 1). Note prolonged venous and sinusoidal opacification in runs #5 and #6, and prominent hepatic vein in run #5.

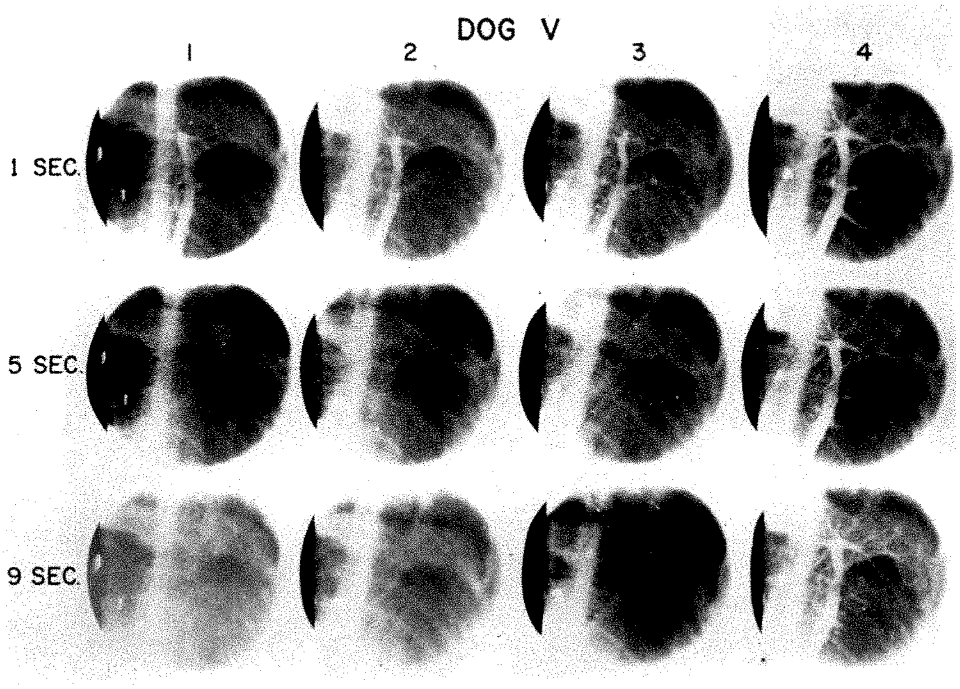

FIG. 3. (Dog V; normal animal.) Individual cine frames exposed 1, 5 and 9 seconds following injections. Run #1: baseline; run #2: following a 30 minute infusion of 100 ml. of 5 per cent casein hydrolysate; run #3:

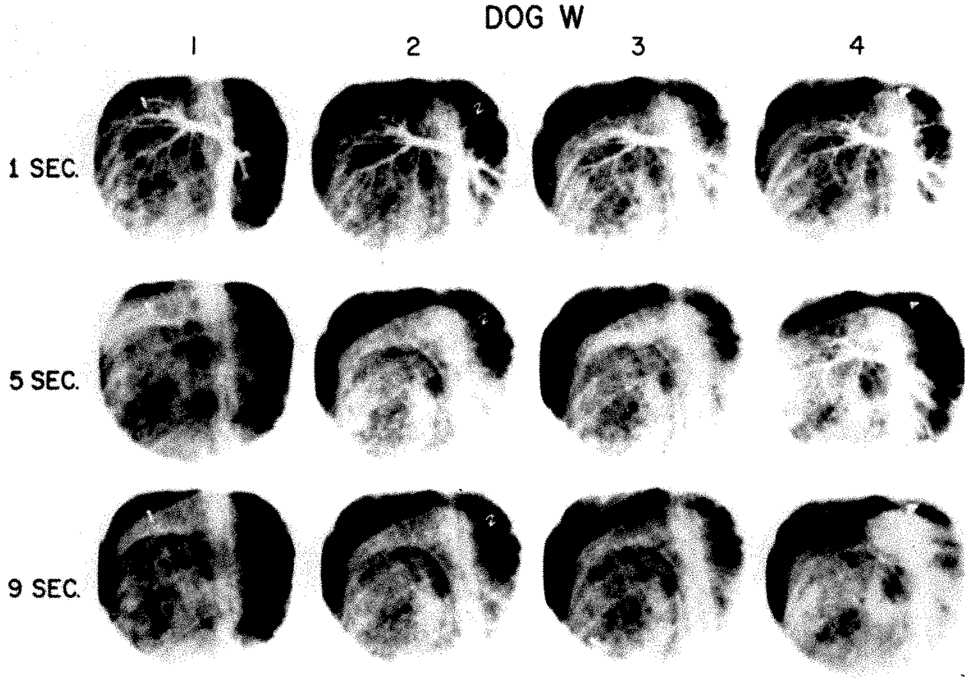

Fig. 4. (Dog W; normal animal.) Run #1: note patchy but relatively prolonged sinusoidal perfusion on baseline cine; run #2: perfusion is increased but still patchy following a 30 minute infusion of 90 ml. of 20 per cent casein hydrolysate; run #3: made 1.5 minutes after epinephrine administration, shows less intense, shortened perfusion and slight narrowing of portal branches; run #4: made after continuation of the infusion and administration of pitressin 1.5 minutes earlier, shows that maximum sinusoidal perfusion is attained but is not entirely confluent.

the 1 moderately cirrhotic animal, stunting and diminished arborization of the intrahepatic portal radicles were noted; vascular morphology was normal in the 3 mildly cirrhotic animals. Variations in the extent of sinusoidal perfusion were usually evident; in general, the greatest parenchymal opacification occurred about the major portal branches, producing a nimbus or halo effect.

Histamine base and pitressin, particularly the latter, produced a definite increase in portal caliber and considerable prolongation of sinusoidal perfusion. The hepatic veins and the vena cava were most frequently seen following administration of pitressin. Scalloping of the hepatic veins was not observed following histamine base infusion. Acetylcholine had no visible effect upon portal venous caliber or upon the intensity and duration of sinusoidal perfusion. Epinephrine and norepinephrine caused a slight reduction in portal venous caliber, decreased perfusion and shortened portal clearance time. The administration of sulfobromophthalein and of casein hydrolysate usually was followed by increased extent, intensity and duration of sinusoidal perfusion. It was not possible to differentiate the 3 mildly cirrhotic animals on the basis of visible reactions to the pharmacologic stimuli. However, in the 4th

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made 4.5 minutes after #2, was preceded by epinephrine and shows diminished caliber and arborization; run #4: made 1.5 minutes after pitressin administration, demonstrates increased caliber, reduced portal flow and prolonged sinusoidal perfusion.

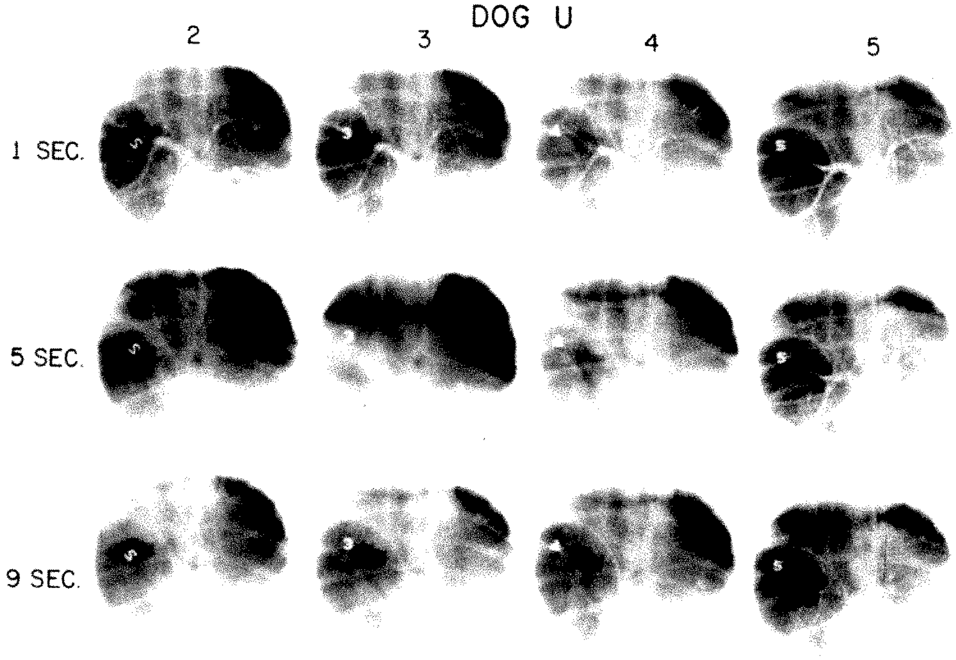
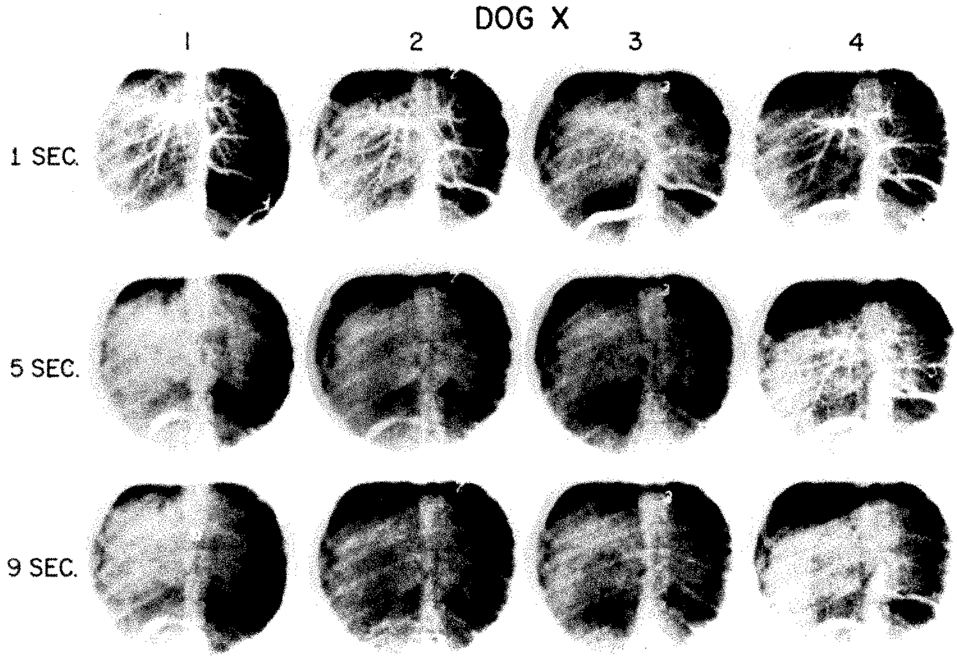


FIG. 5. (Dog U; cirrhosis, moderately severe.) This animal's stomach contained food administered 23 hours earlier. Run #2: baseline study showing attenuation and diminished arborization of portal branches; run #3: made at the end of a 20 minute infusion of sulfobromophthalein, shows that sinusoidal perfusion is slightly increased; run #4: following epinephrine, demonstrates portal venous narrowing, increased flow and reduced perfusion; run #5: following pitressin, shows stagnant portal flow.



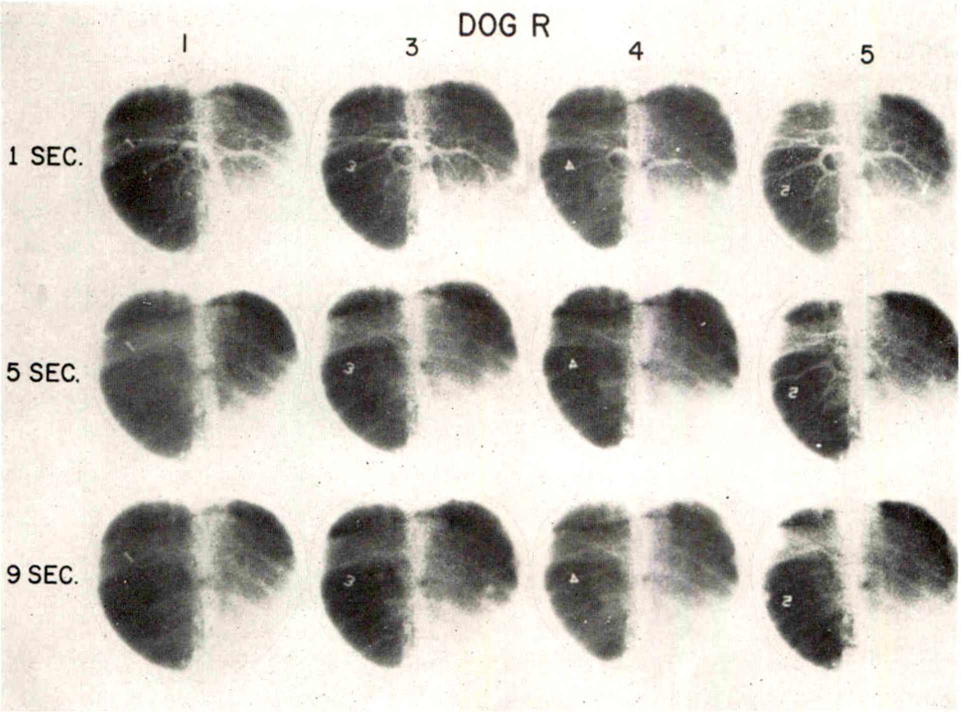


Fig. 7. (Dog R; cirrhosis, slight.) Run #1: baseline; run #3: after histamine infusion, shows slightly increased venous caliber and sinusoidal perfusion; run #4: following epinephrine, shows reduced venous caliber and sinusoidal perfusion; run #5: after pitressin, maximal perfusion together with stagnation of contrast material is noted. In this animal progressively increasing hepatic opacification, due to uptake of the contrast material, was observed.

cirrhotic animal, sinusoidal perfusion was always less intense, regardless of the stimulating agent administered, than in the normal animal.

B. *Quantitative Measurements.* The portal venous clearance time was arbitrarily defined as the time required for the radiopaque bolus to pass the portal bifurcation. In the normal anesthetized animal, the baseline portal venous clearance time was 3.1–3.8 seconds. In 3 of the 4 cirrhotic animals the clearance times fell within this range; in the 4th, it was reduced to 2.3 seconds. There was no significant change in clearance time after administration of acetylcholine. Following epinephrine administration, clearance time was reduced to a range of 2.1–3.0 seconds. The effect of norepinephrine was similar. The infusion of histamine base prolonged portal venous clearance time, which rose to a range of 4.7–5.9 seconds. The clearance time was elevated to a range of 7.4–10.6 seconds following pitressin administration. The infusions of sulfobromophthalein or casein hydrolysate had variable effects; in some

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Fig. 6. (Dog X; cirrhosis, slight.) Run #1: baseline; run #2: following infusion of 100 ml. of 20 per cent casein hydrolysate in 33 minutes, shows that perfusion is not significantly altered; run #3: following epinephrine, shows definite reduction of opacified portal branches but little change in perfusion; run #4: after administration of additional casein hydrolysate and 1.5 minutes after pitressin, demonstrates intravenous stagnation and intense, prolonged sinusoidal perfusion.

animals there was no change in clearance time, but in others this decreased by amounts ranging up to 0.6 seconds. This decrease cannot necessarily be considered significant.

The administration of double doses of the pharmacologic agents to 3 normal animals did not further alter portal clearance times.

Measurements of portacaval circulation time were not possible because of the typically indistinct onset of caval opacification. As indicated above, measurements of perfusion changes in various portions of the hepatic parenchyma were not reproducible. However, in any given set of measurements of perfusion, increased intensity and duration of sinusoidal opacification were demonstrable following administration of histamine base or pitressin (Fig. 9). Superimposed upon these photometric curves were fluctuations corresponding to the respiratory excursions of the diaphragm.

Intermittent manometric recordings of portal pressure were employed primarily for determining the end-point of hepatic circulatory changes caused by the various administered agents. It was considered that, other factors being constant, the return of altered portal pressure to baseline levels was a reasonable indication of termination of drug effect. It was noted that portal pressure rose following histamine base and epinephrine and fell following vasopressine. Acetylcholine, sulfobromophthalein and casein hydrolysate did not significantly alter portal pressure.

DISCUSSION

Cinefluorography is a useful method of investigating certain dynamic aspects of the portal and hepatic sinusoidal circulation in large animals. This method permits demonstration of changes in portal venous caliber, intrahepatic portal arborization, portal venous flow rates and parenchymal sinusoidal perfusion in the normal and damaged liver. By providing a permanent visual record of these rapidly occurring hemodynamic events, cinefluorography affords a unique means for their conceptualization.

In this investigation variables were elim-

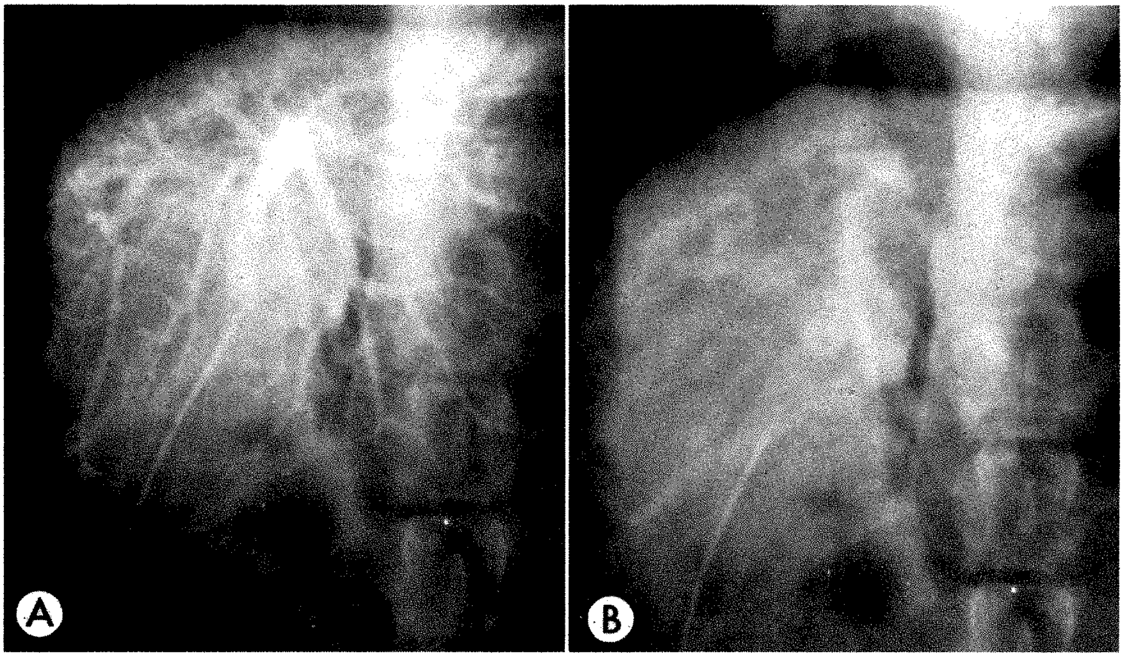

FIG. 8. Early (*A*) and late (*B*) roentgenograms from serial roentgenographic portography made with rapid film changing device. The fine portal radicles are clearly delineated in *A*, while in *B* parenchymal opacification is slightly increased and the vena cava is visible above the diaphragm.

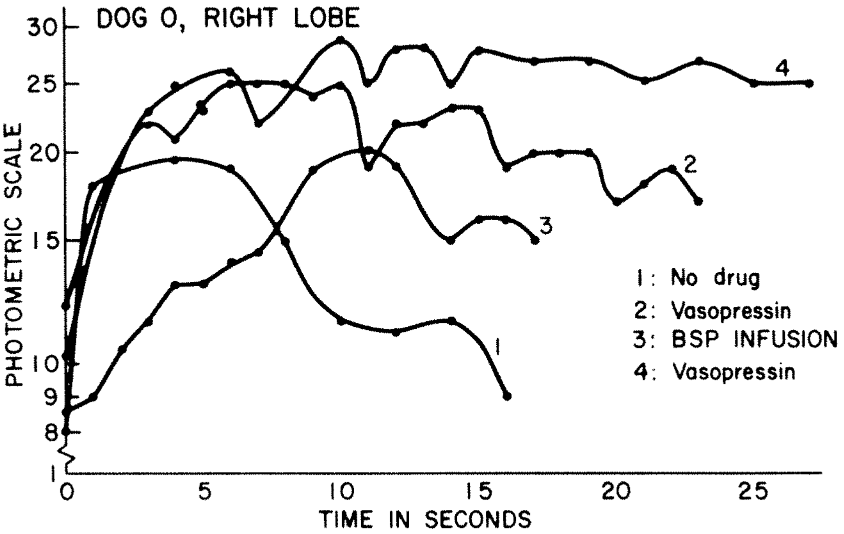

Fig. 9. (Dog O; normal animal.) Photodensitometric tracings of hepatic parenchymal density following administration of various agents. Respiratory fluctuations are superimposed upon some of these curves. Although not reproducible on a point-for-point basis, these tracings confirm the observation that maximal intensity and prolongation of sinusoidal perfusion follow the administration of pitressin (vasopressin).

inated insofar as possible. The surgical technique of catheter implantation was standardized. Animals were examined in the fasting state. There was no trauma to the liver. The injected volume of contrast material and the doses of the pharmacologic agents were determined on a per kilogram basis. For a given animal, the speed and pressure of injection of contrast material, cine exposure factors, the target-screen distance, the position of the animal and the cine developing factors were held constant. However, respiratory amplitude and rate were not controlled, and film artifacts were occasionally encountered.

The observed changes in intrahepatic portal and sinusoidal blood flow conformed in general to those anticipated on the basis of the extensive literature, recently summarized by Bradley[2] and Green and Kepchar,[8] dealing with the effects of these agents. Of interest, however, was the invariable marked slowing of portal venous flow associated with increased caliber and prolonged sinusoidal perfusion that followed administration of pitressin. This striking effect was demonstrable only 90 seconds after intraportal administration of the hormone, and suggests the possibility of significant local action of pitressin upon the hepatic circulation. The lack of hepatic circulatory alteration after administration of acetylcholine agrees with the observation that intraportal administration of this agent has little or no effect at dosage levels usually employed.[2]

In the fasting, anesthetized state, the extent of sinusoidal perfusion was nonuniform. Maximum opacification occurred in those sinusoids closest to the major portal branches. This finding would appear to be in keeping with the concept of restricted intrahepatic flow demonstrated in small animals.[5] The infusions of sulfobromophthalein and casein hydrolysate, comprising a metabolic "load" upon hepatic parenchymal cells, were administered to increase the extent and volume of sinusoidal perfusion. It has been demonstrated that oral feedings of homogenized lean meat are followed by elevation of estimated hepatic blood flow and of wedged hepatic venous pressure in humans.[4] In the present investigation, both types of infusion were usually followed by more intensive and extensive sinusoidal opacification but, as noted, such changes could not be quantitated.

Induced cirrhosis was moderately severe in 1 animal and mild in 3. In the latter, the hepatic circulatory responses did not differ discernibly from those noted in the normal canine liver. In the former animal, which exhibited the characteristic vascular morphologic changes of cirrhosis, the circulatory responses appeared to differ quantitatively but not qualitatively.

Hepatic accumulation of iodinated roentgenographic contrast agents has been described.[10] Increased opacity of the hepatic parenchyma following multiple injections of hypaque was observed in several of the animals in this investigation, but was pronounced in none.

A disadvantage of cineportography is the relatively unsharp delineation of morphologic features. Preliminary studies leading to this investigation were performed with a serial film-changing device which produced clearer images of major vascular structures and regularly demonstrated the efferent hepatic blood flow. Larger cine film size and particularly ultra-fine grain emulsion should improve resolution of the cineportogram and perhaps permit accurate measurement of vessel caliber.

It is reasonable to predict that additional data can be derived from cinefluorographic study of the hepatic circulation. With refinement of photometric techniques, variations in the extent of regional sinusoidal perfusion should be susceptible of quantitation, and portal flow rates more accurately measured. Such data would have considerable physiologic significance. In view of the effect of diaphragmatic position and tone upon hepatic venous blood flow, it appears that apnea should be induced for the duration of each cine sequence.

Due to the presence in the dog of muscular hepatic veins that comprise a "throttling mechanism" capable of blocking venous drainage,[1,9] extrapolation of the results of this investigation to the human liver must be made with caution. Nevertheless, it would be of interest to learn whether prolonged opacification of human hepatic sinusoids might be achieved by means of imposition of a metabolic "load" to increase perfusion, administration of pitressin to slow hepatofugal flow, and intrasplenic injection of contrast agent. In the event that prolonged uniform opacification was produced without significant side effects such as systemic hypertension, the radiopaque liver could then be examined by multiple simultaneous laminagrams. In this manner small, deeply situated space-occupying lesions—such as are currently not demonstrable by other methods—might be detected.

SUMMARY

Portal venous flow and intrahepatic sinusoidal perfusion were investigated by cinefluorography under standardized conditions in 13 normal and 4 cirrhotic dogs. Hepatic circulatory changes produced by several pharmacologic agents were observed and analyzed. Pitressin produced significant stagnation of portal flow and sinusoidal perfusion 1.5 minutes after intraportal administration. A similar alteration in flow took place after histamine administration, although a hepatic venous throttling mechanism was not demonstrated. Cineportography is a useful method for qualitative investigation of the hepatic circulation, with potentialities for quantitative photometric analysis.

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HAMARTOMA OF THE SPLEEN*

ANGIOGRAPHIC OBSERVATIONS

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HAMARTOMA of the spleen is a rare lesion that to our knowledge has never been diagnosed preoperatively. Of the 38 cases reported in the literature,[2,7,8,11,13,14,15] symptoms were present in only 7. In 1 patient the lesion was discovered at surgical exploration for a traumatic rupture of the spleen. Hypersplenism was a prominent clinical feature in the cases reported by Videbaek,[14] and Schrijver and Verdonk.[11] Five patients presented with complaints related to a left upper quadrant abdominal mass. In the remainder, the patients had been asymptomatic and the lesions were found incidentally during routine postmortem examination.

Hamartoma of the spleen has no sex predilection and has been reported in all age groups, including 2 children under 5 years of age. The tumors are benign, varying in size from several millimeters to 11 cm. in diameter. They are spherical, well defined and demarcated, but not truly encapsulated. Most often the lesions are solitary, although 3 instances of multiple nodules have been described. The tumors are confined by the normal splenic capsule.

Microscopically, the tumor is composed of splenic pulp tissue with absence of normal trabeculae and follicles. There is often considerable fibrosis present. Compressed trabeculae at the border of the hamartoma may simulate a capsule. Typically, large, thin walled, endothelial lined vascular spaces are present.[14] Calcification is rare.

The roentgenographic features of a case which we have observed were of sufficient interest to warrant reporting, particularly since no description of the angiographic appearance has been previously published.

REPORT OF A CASE

B.D., a 4 year old white boy, was referred to the Stanford Medical Center 2 months after an abdominal mass was discovered during a routine physical examination. The child had been well prior to admission. There was no history of trauma, fatigue, fever, easy bruising or jaundice. There was no known exposure to infectious or parasitic agents. Other elements in the past history were not relevant.

On physical examination, the boy appeared healthy and well developed. Positive findings were limited to the abdomen. An 8×12 cm. hard, nontender mass was felt in the left flank. This was thought to be spleen and a splenic notch was palpable. The liver edge was 2 cm. below the right costal margin.

Laboratory studies were normal except for moderate anemia and persistent proteinuria.*

Roentgenologic Examination. Chest roentgenograms and skeletal survey were normal. Excretory urograms (Fig. 1, *A* and *B*) demonstrated an extrarenal mass which displaced the left kidney medially and superiorly, producing slight deformity of the lower pole calyces. The mass impinged upon and displaced the posterior wall of the stomach. It appeared circular and contained no calcification. The psoas margin was obliterated.

An upper gastrointestinal series disclosed no intrinsic abnormalities. The small bowel and splenic flexure were mobile and displaced medially by the mass.

Abdominal aortography was performed under local anesthesia by the percutaneous Seldinger technique.[12] A PE No. 205 catheter was passed into the aorta from the right femoral artery. With the tip of the catheter at the level of the first lumbar vertebra, two injections of 10 cc. of 76 per cent renografin were made, with serial

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* This aspect of the case will be discussed at length in a separate communication.[9]

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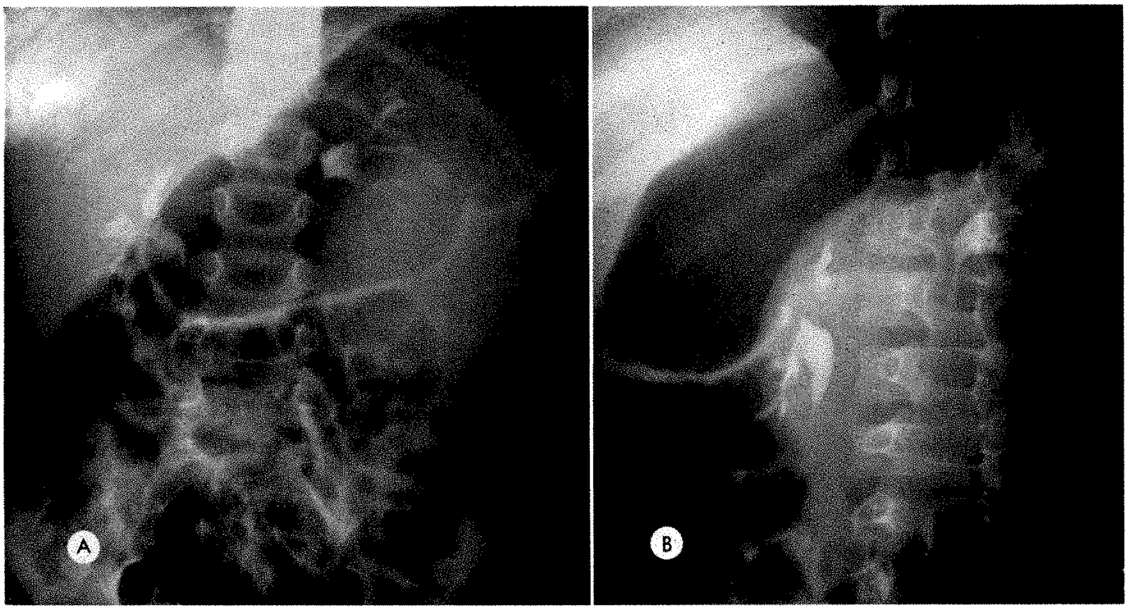

Fig. 1. (*A*) Supine and (*B*) right posterior oblique excretory urograms. The stomach has been distended with a carbonated beverage. The round soft tissue mass in the left upper quadrant cannot be separated from the splenic shadow. There is no calcification within the mass. The left kidney is displaced medially and superiorly with slight distortion of the lower pole calyces. The posterior wall of the stomach is impinged upon and the gas filled loops of small bowel and the splenic flexure are displaced inferiorly and medially. An upper gastrointestinal series confirmed the findings on plain roentgenograms and showed no mucosal involvement.

filming in the anteroposterior and left posterior oblique projections (Fig. 2, *A* and *B*). The branches of the splenic artery were splayed around a tumor mass which occupied approximately one-half of the splenic substance. Normal splenic branches were present in the lower pole and superior one-third of the spleen. In the early arterial phase, numerous large, tortuous vessels were visualized within the tumor mass. There was filling of venous structures in less than 1/6th of a second. These vascular lakes remained filled with contrast material in the late arterial and capillary phases (Fig. 2*C*).

The left renal artery was displaced superiorly and medially and the lower pole vessels were straightened and stretched due to extrinsic compression. The jejunal and middle colic branches of the superior mesenteric artery were displaced inferiorly and mesially. The splenic vein was patent. A diagnosis of congenital angiomatous malformation (hemangioma) or hemangiosarcoma was suggested.

Hospital Course. At exploratory laparotomy, a large firm mass was palpable in the spleen and total splenectomy was performed (Fig. 3). The abdominal viscera were normal but displaced by the mass. The left renal vein appeared to be compressed. The patient recovered uneventfully and was discharged on the tenth postoperative day.

Pathology. The spleen weighed 300 gm. and contained a well delimited mass 7 cm. in diameter with cystic spaces and fibrinous septa (Fig. 3). Microscopic sections (Fig. 4) of the mass revealed it to be composed predominantly of splenic pulp cells and sinuses but free of normal trabeculae and lymphoid follicles. Many large and cavernous vascular channels were scattered throughout the tumor. The border of the lesion was composed of compressed adjacent splenic tissue and nonconnecting trabeculae without a true capsule. The appearance was considered characteristic of a hamartoma.

DISCUSSION

The diagnosis in this patient was of particular interest because of the roentgen features of the tumor, and its effects on adjacent structures.

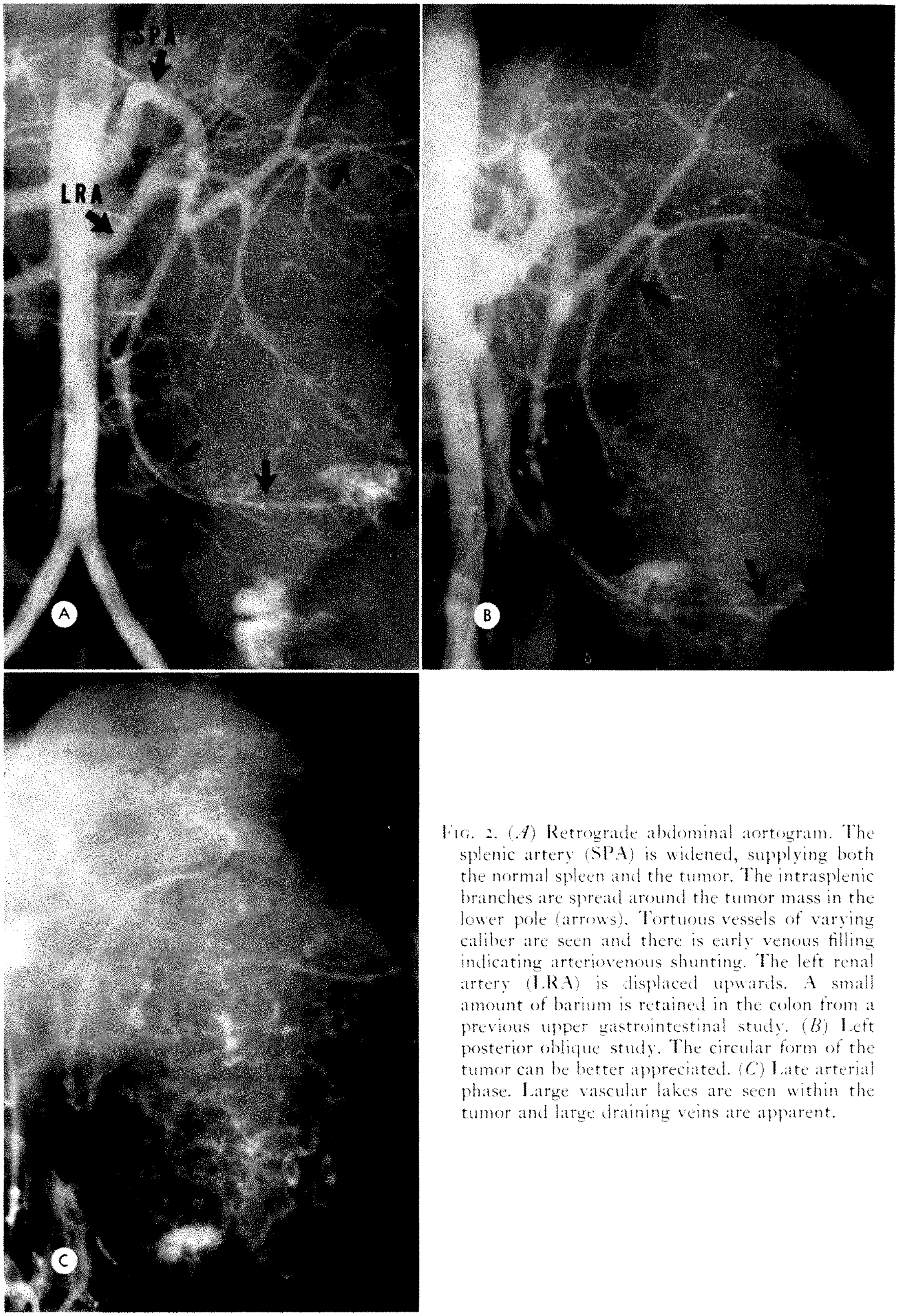

FIG. 2. (*A*) Retrograde abdominal aortogram. The splenic artery (SPA) is widened, supplying both the normal spleen and the tumor. The intrasplenic branches are spread around the tumor mass in the lower pole (arrows). Tortuous vessels of varying caliber are seen and there is early venous filling indicating arteriovenous shunting. The left renal artery (LRA) is displaced upwards. A small amount of barium is retained in the colon from a previous upper gastrointestinal study. (*B*) Left posterior oblique study. The circular form of the tumor can be better appreciated. (*C*) Late arterial phase. Large vascular lakes are seen within the tumor and large draining veins are apparent.

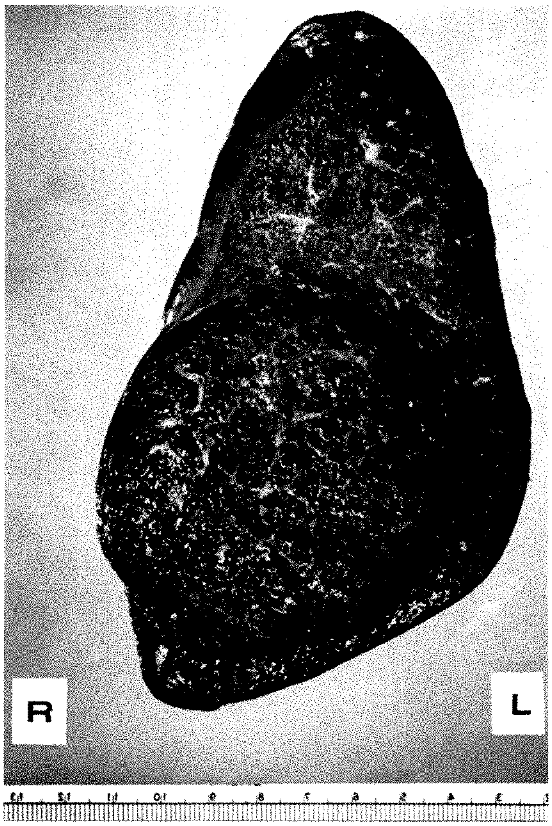

Fig. 3. Cut surface of gross specimen. The tumor mass in the lower pole is distinct from normal tissue which surrounds it. Large fibrous septa and vascular lakes can be identified.

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The kidney was displaced medially and superiorly. Accompanying this displacement, the renal vein was compressed, producing proteinuria, as in renal-vein thrombosis, which disappeared after surgery. The character of the displacement by the enlarged spleen was unusual, since more often the kidney will be pushed medially and caudad.[5] The explanation lies in the location of the tumor in the lower pole of the spleen, which was enlarged far more than the upper portion of the spleen. The fact that an intraperitoneal structure was able to affect the retroperitoneal viscera has been thoroughly recognized but deserves reemphasis. The effect on the stomach, small

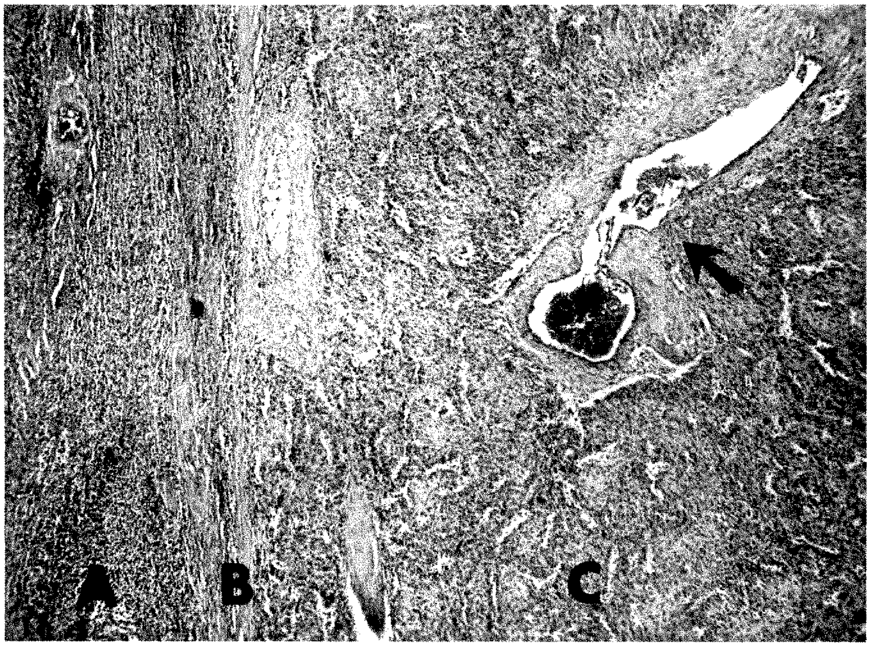

Fig. 4. Microscopic section, ×63. Normal splenic tissue (A) is seen on the left. A border of compressed splenic parenchyma (B) separates the hamartoma (C) from normal tissue. A large vascular lake (arrow) is seen in this photomicrograph. The tumor is composed of the same elements present in the adjacent normal splenic tissue.

bowel, and colon was typical of that found whenever the spleen enlarges.

The appearance of the splenic vessels on arteriography strongly suggested the possibility of a malignant lesion. The vessels were tortuous, ramified irregularly, communicated directly with veins, and were relatively large in size. They resembled the neovasculature of malignant tumors of the kidney and other viscera, and prompted serious thought as to the presence of an angiosarcoma.

The tumor vessels are, in fact, primitive in appearance and plexoid in character. A rest tumor, such as a hamartoma, characteristically contains primitive and embryonic constituents of the organ in which it is found, and the vasculature clearly reflects this. It would be impossible to distinguish these vessels in the spleen, therefore, from the malignant vessels seen in tumors of the kidney, liver, bladder and brain.

DIFFERENTIAL DIAGNOSIS[1,6]

In considering the causes for splenic enlargement, one must first exclude systemic conditions that affect the spleen. Infections, lymphoma, leukemia and the reticuloses are the common disorders in this category. They can be diagnosed on the basis of clinical findings as well as roentgenographic involvement of other systems, commonly the long bones and lymph nodes.

Other primary neoplasms of the spleen include dermoid and epidermoid cyst, lymphangioma, hemangioma, serous cyst and polycystic disease. The most common cause of splenic cyst is echinococcus disease, prior trauma accounting for the majority of nonparasitic cysts. Peripheral calcification may be present in the wall of the cyst. Cystic fluid may be obtained by percutaneous puncture, as has been demonstrated inadvertently during attempted splenoportography.[3,4]

The angiographic characteristics of splenic lesions have been described by Rösch and Bret.[10] Cystic lesions of the spleen appear similar to cystic lesions of other organs, causing compression and displacement of surrounding normal parenchyma. Infiltrative diseases such as leukemia and lymphoma stretch and narrow the intrasplenic vessels. No prior description of the angiographic appearance of a vascular splenic tumor has been found in the literature.

The left kidney, pancreas, left liver lobe, retroperitoneum, omentum and mesentery are the primary sites of origin of masses extrinsic to the spleen which enter into the differential diagnosis. Aneurysms of the aorta and its branches are easily excluded by aortography. If the routine roentgenographic studies of the urinary and gastrointestinal tracts are inconclusive in localizing the mass, aortography and selective arteriography may be definitive.

SUMMARY

Hamartoma of the spleen is a highly vascular tumor, composed of splenic pulp tissue without trabeculae and follicles. Angiographic study of such a tumor has demonstrated large, plexoid vessels, resembling the neovasculature of certain malignant tumors in adults. The pathologic findings and differential diagnostic considerations are discussed.

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RADIOISOTOPE PHOTOSCANNING IN THE DIAGNOSIS OF PANCREATIC DISEASE*

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A NEED has long existed in clinical medicine for improvement in diagnostic methodology for visualizing the pancreas and detecting disease within it. The high rate of pancreatic uptake of amino acids has been recognized for some time.[6] This uptake is a reflection of the rapid rate of synthesis of the digestive enzymes. From 5 to 10 per cent of most amino acids are found in the pancreas within an hour after administration. Unfortunately, none of the elements in the structure of amino acids (C, H, N, O and S) has gamma ray-emitting isotopes with properties suitable for scanning. Externally labelled amino acids (*e.g.* I^{131}-iodotyrosine), because of their inability to take part in protein synthesis, show no pancreatic uptake. The chemical similarity of selenium and sulfur is such that the substitution of Se^{75} for sulfur within a methionine molecule results in an analogue which has the necessary biologic properties of the natural amino acid.[2,5] In 1961, Blau and Manske demonstrated[2] that Se^{75}-selenomethionine is concentrated in the pancreas in an amount (gram for gram) about 8 to 9 times that found in the liver. Blau and Bender[1] have been able to visualize the pancreas by isotope photoscanning in approximately two-thirds of a group of 50 patients without known pancreatic disease after intravenous administration of 3 to 3.5 μc/kg. body weight of Se^{75}-selenomethionine. The estimated total body dose to the patient is about 0.6 rad, delivered over several months.[1] Two to three hours after a high protein breakfast, the pancreas is stimulated to secrete its preformed "cold" proteins by administration of a mixture of cholecystokinin and pancreozymin (Cecekin Vitrum).* One hour later Se^{75}-selenomethionine is injected for incorporation into the rapidly reforming protein secretions of the pancreas.

MATERIALS AND METHODS

SELECTION OF PATIENTS

Patients with no known pancreatic disease ("controls") and those with suspect pancreatic malignancy or chronic pancreatitis were referred to the Radioisotope Laboratory for pancreatic scanning. Thirty-two patients have been studied to date and form the basis of this report.

PERFORMANCE OF SCANS

Photoscans were performed using the technique of Blau and Bender[1] described above. Se^{75}-selenomethionine† in a dose of 250 μc was given intravenously 1 hour after the Cecekin, and the scan was performed 30 minutes after the administration of the radioactive tracer.

A liver scan using I^{131}-rose bengal was performed 48 to 72 hours prior to the pancreatic photoscan to demarcate the liver and, in particular, the lower borders of the liver. The hepatoscan was then employed to facilitate delineation, where feasible, of pancreas from liver on the Se^{75} scan. A Picker Magnascanner‡ was employed for all photoscans.

RESULTS

ILLUSTRATIVE CASES

Normal Pancreas. Figure 1, *A* and *B* shows a

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* Apoteksvarucentralen Vitrum Company, Box 12170, Stockholm 12, Sweden.

† Squibb Laboratories, New Brunswick, New Jersey.

‡ Picker X-Ray Corporation, White Plains, New Jersey.

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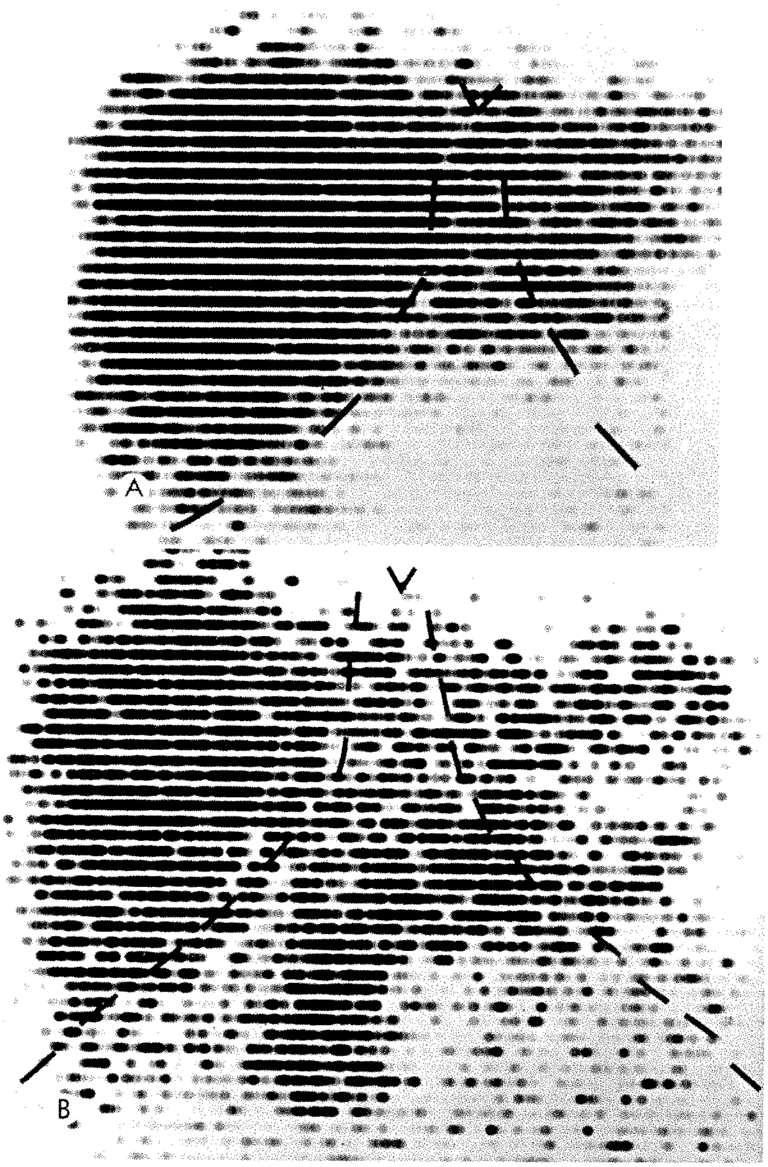

Fig. 1. (*A*) Normal liver scan performed with I^{131}-rose bengal in a patient with no known hepatic or pancreatic disease. (*B*) Normal pancreatic scan performed with Se^{75}-selenomethionine in the same patient outlining the liver and pancreas with adequate separation of the two organs.

liver scan with I^{131}-rose bengal and a pancreatic scan with Se^{75}-selenomethionine in a patient with no known hepatic or pancreatic disease. The scans demonstrate a normal liver and good visualization of the entire pancreas with adequate separation of the two organs on the Se^{75} photoscan.

Pancreatic Neoplasms. Figure 2 shows a pancreatic photoscan in a patient with a documented ulcerogenic non-beta islet cell carcinoma of the pancreas. This case has been reported in detail elsewhere.[3]

Figure 3, *A* and *B* depicts the hepatic and pancreatic scans obtained in a 66 year old male admitted with a 6 month history of weakness, weight loss and painless jaundice. The scans were interpreted as showing intrahepatic metastatic disease, with a lesion in the head of the pancreas. At laparotomy an adenocarcinoma of the head of the pancreas was found in the "cold" area indicated on the photoscan with metastases to the liver in the sites noted on the initial hepatoscan.

Pancreatitis. The pancreatic photoscan of a

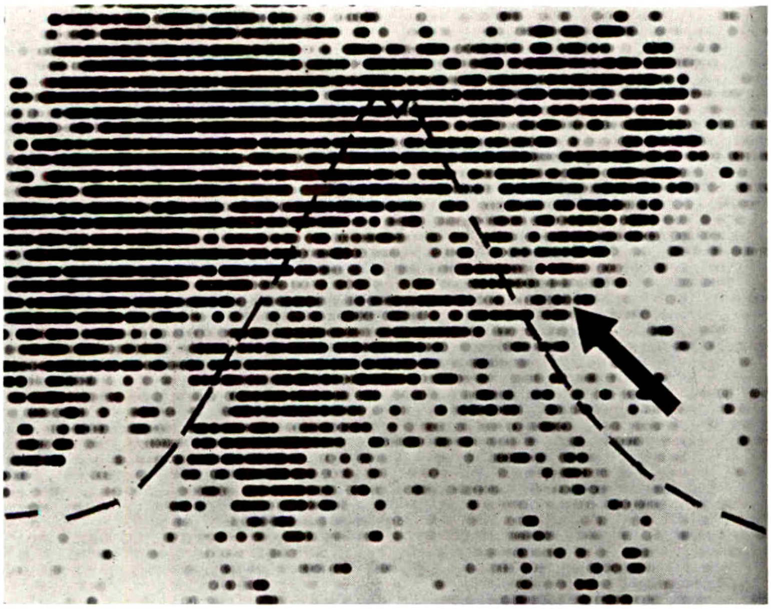

Fig. 2. Pancreatic photoscan in a patient with an ulcerogenic non-beta islet cell carcinoma. The arrow indicates the "cold" area within the mid-portion of the body of the pancreas where the tumor was found. (Reproduced with permission of *Gastroenterology*[3] and Williams & Wilkins Co.)

53 year old male with well documented chronic pancreatitis is shown in Figure 4. This scan demonstrates the typical marked decrease in uptake of radioselenium throughout the pancreas most often observed in this condition.

TOTAL RESULTS

To date, we have performed 32 pancreatic scans using this technique. As can be seen from Table 1, we have achieved adequate visualization of the pancreas by scanning with Se^{75} in only 18 of these patients. The obvious difficulty is that, although on a gram for gram basis the pancreatic uptake of Se^{75}-selenomethionine ex-

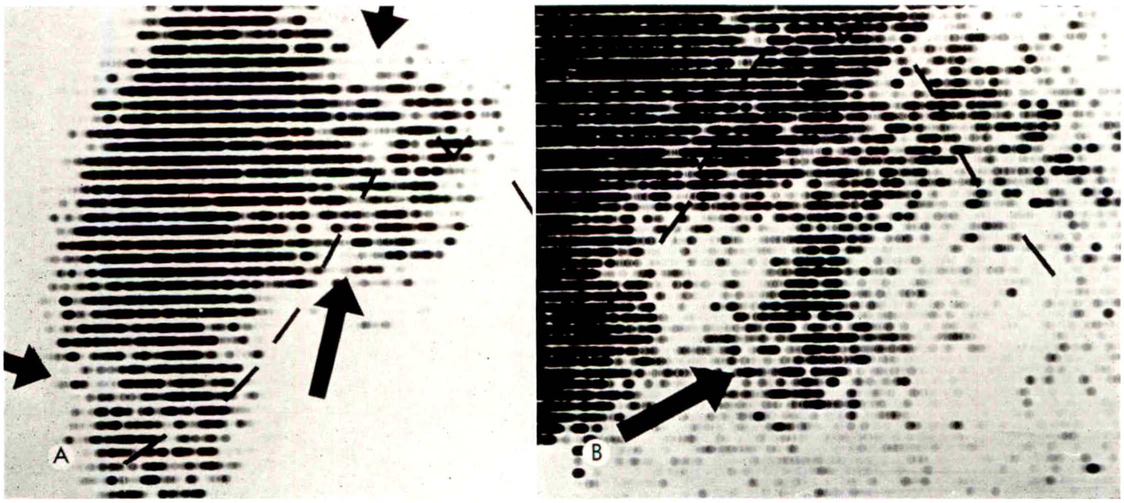

Fig. 3. (*A*) Liver scan in a patient with an adenocarcinoma of the head of the pancreas. The several areas of decreased isotope uptake within the liver (arrows) suggest extensive metastatic disease. (*B*) Pancreatic scan on the same patient. The arrow indicates the "cold" area in the head of the pancreas where the tumor was found.

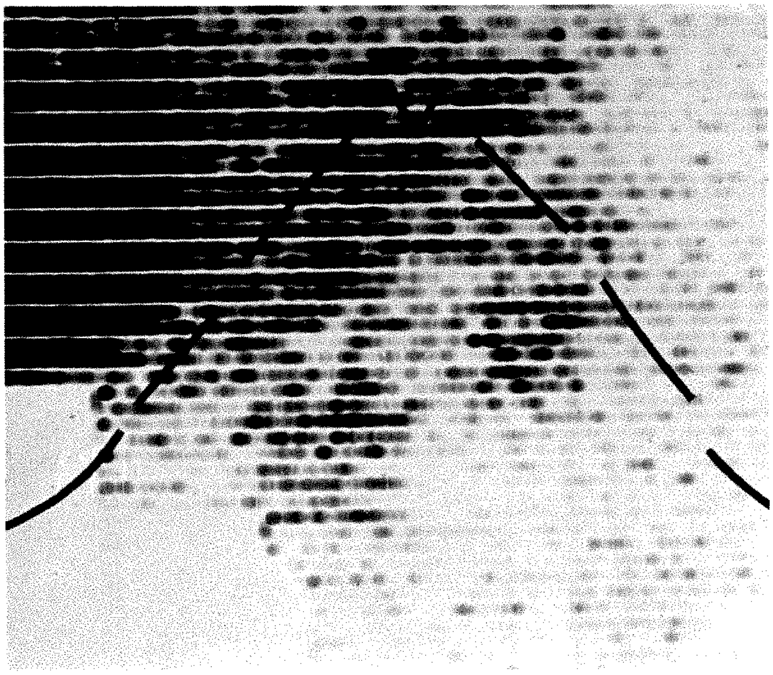

FIG. 4. Pancreatic photoscan in a patient with chronic pancreatitis. Note the marked decrease in isotope uptake throughout the organ and the generally spotty distribution of radioactivity.

ceeds that of the liver, the relative sizes of the organs are such that the *total* radioactivity in the overlying liver exceeds that in the pancreas. It follows, therefore, that adequate delineation of the pancreas with this technique is possible only where there is sufficient anatomic separation of the pancreas from the liver for separate projection on the photoscan. The diagnostic limitations engendered by organ "overlap" are illustrated in Figure 5. These difficulties notwithstanding, we have been able to demonstrate the presence of a "cold" area in 3 of 4 confirmed cases of pancreatic carcinoma and in both of the 2 presumptive cases.

ATTEMPTED BLOCKADE OF HEPATIC UPTAKE OF Se^{75}-SELENOMETHIONINE

An attempt was made to overcome the problem of organ "overlap" by administration of large doses of nonradioactive methionine to a series of patients prior to the performance of the pancreatic scan. It was felt that this prior "saturation" of the liver with "cold" methionine might, to some degree, inhibit the hepatic uptake of radioactive selenomethionine at the time of pancreatic scanning. Since the discharge of preformed protein secretions from the pancreas had been achieved by the administration of Cecekin prior to the injection of

TABLE I

RADIOISOTOPE PHOTOSCANNING OF THE PANCREAS

Patient Group	Photoscan	
	Visualization	Nonvisualization
Controls*	11	13
Pancreatic Carcinoma (confirmed)	3	1
Pancreatic Carcinoma (presumptive)†	2	0
Pancreatitis	2	0
Totals	18	14

* No known pancreatic disease.
† No tissue diagnosis.

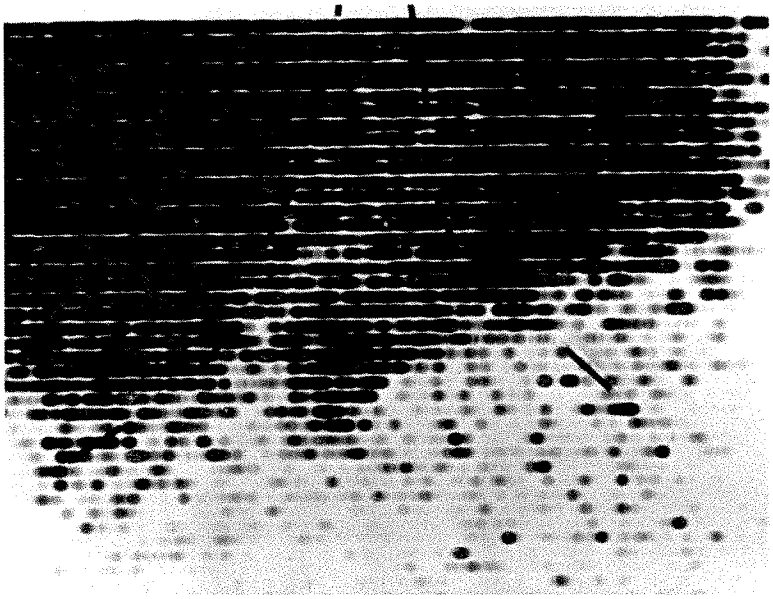

FIG. 5. Pancreatic photoscan illustrating the problem of organ "overlap." Only the head of the pancreas is clearly demarcated; the remainder of the organ is obscured by the overlying left lobe of the liver.

Se^{75}-tagged methionine, pretreatment with nonradioactive methionine might not affect pancreatic uptake of the labelled amino acid. The net result would be a greater relative concentration of Se^{75}-selenomethionine in the pancreas, facilitating delineation from the overlying liver.

Methionine, 12 gm. daily in divided doses, was administered to 6 patients for a period of 5 to 7 days prior to the performance of the pancreatic scan. It was found, however, that this approach did not materially affect the hepatic uptake of Se^{75}-selenomethionine in any of the patients studied and offered no solution to the problem of organ "overlap."

DISCUSSION

Based on the results of this initial experience, it is our feeling that pancreatic photoscanning with Se^{75}-selenomethionine is a useful additional procedure in the diagnosis of pancreatic disease. However, it is apparent that the limitations of the technique are considerable since we were able to obtain technically satisfactory scans in only 18 of the 32 patients studied. [Haynie *et al.*[4] have recently reported adequate studies in 37 of 58 patients scanned, utilizing the same technique]. Aside from the frequent inability to delineate pancreas from liver because of hepatic enlargement or downward displacement of the liver alluded to above, it is apparent from our study there is normally a great variability from patient to patient in the size and configuration of the pancreas and in the pattern of distribution of radioactivity. It is obvious, therefore, that small lesions, particularly those located at the periphery of the pancreas, will, as a rule, not be detected and that a negative scan does not exclude pancreatic disease. While more satisfactory visualization of this organ must await the development of radioactive tracers with greater and/or more selective pancreatic specificity, the present technique does offer a relatively simple method of visualizing the pancreas without surgical intervention.

SUMMARY AND CONCLUSIONS

The results of pancreatic photoscans performed with Se^{75}-selenomethionine in 32 patients have been reviewed in order to evaluate the reliability and usefulness of this procedure in the diagnosis of pancreatic disease.

Adequate visualization of the pancreas was obtained in somewhat more than one-half of the patients studied, and the photoscan proved diagnostic in 3 of 4 surgically confirmed cases of pancreatic carcinoma and in 2 cases of well-documented chronic pancreatitis. While the limitations of the procedure are considerable, it offers a simple method of pancreatic visualization without resort to surgery and merits further study.

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PORTAL VEIN GAS: A GRAVE PROGNOSTIC SIGN*

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THE roentgenographic diagnosis of gas in the portal venous system is readily made when one is familiar with its appearance. The characteristic finding is a pattern of linear branching gas shadows in the right upper quadrant radiating outward to terminate near the periphery of the liver. This pathognomonic appearance allows differentiation from gas in the biliary tree, with which it is most often confused. Portal vein gas was first reported in 1955 when Wolfe and Evans[5] described 6 cases occurring in infants, and 10 additional cases have been reported since then. Gas in the portal venous system indicates a grave prognosis, and all the reported patients have died shortly after its recognition. Two cases were observed recently in this hospital.

REPORT OF CASES

CASE I. B. G., a 30 year old white woman, was first seen here in June, 1958. In January, 1958, her mesenteric small bowel and proximal colon were resected because of intestinal gangrene secondary to malrotation of the bowel and superior mesenteric artery occlusion. Following this, she was seen on numerous occasions for management of intestinal malabsorption and electrolyte imbalance problems. Her average weight after surgery was 55 pounds.

On May 21, 1962, she entered complaining of weakness and periods of disorientation. Her electrolyte imbalance was corrected, but she developed intermittent abdominal distention, nausea, vomiting and obstipation. On June 11, 1962, she developed severe lower abdominal pain and tachycardia. The abdomen was soft, and the bowel sounds were hypoactive. This was followed by shock, a semicomatose state, and fever. Bloody fluid was obtained from a nasogastric tube. Her condition progressively deteriorated, and she expired 2 days later.

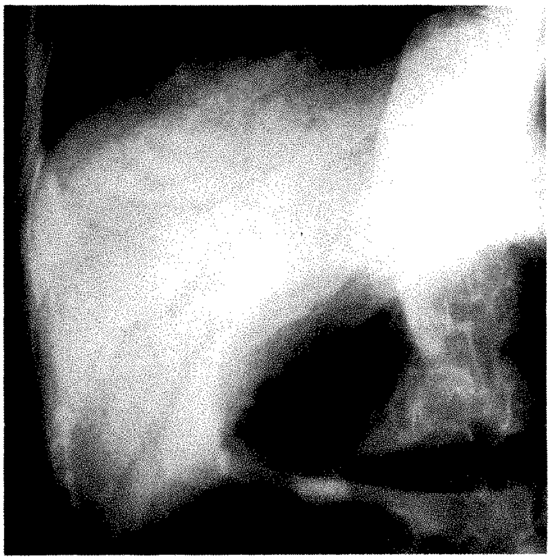

FIG. 1. Case I. Roentgenogram of the right upper abdomen shows the linear branching gas shadows typical of gas in the portal venous system. Note the extension of the arboreal pattern to the periphery of the liver.

Roentgenographic Findings. Roentgenograms of the abdomen shortly after the onset of severe abdominal pain (Fig. 1) revealed an arboreal pattern of radiolucency in the liver area, which had the appearance of gas in the portal venous system. Intestinal distention was noted, but the bowel pattern was not unusual for this patient. The following day, abdominal roentgenograms (Fig. 2) revealed a definite decrease in the amount of gas in the portal venous system. Shortly before death, roentgenograms (Fig. 3) demonstrated complete disappearance of the linear gas pattern in the hepatic area. However, gas was noted in the bowel wall, particularly evident in a segment of the colon in the left upper quadrant.

Autopsy Findings. There was no detectable gas in the portal venous system or biliary tree. Areas of necrosis and ulceration with acute inflammatory reaction, edema, and hemorrhage were noted in the colon. The inferior mesenteric artery was patent. Multiple submucosal and serosal gas cysts were present in the colon, and a large number of gram positive rods was noted in many of these cysts. The organisms morphologically resembled clostridia, and postmortem cultures of the bowel wall grew *Clostridium per-*

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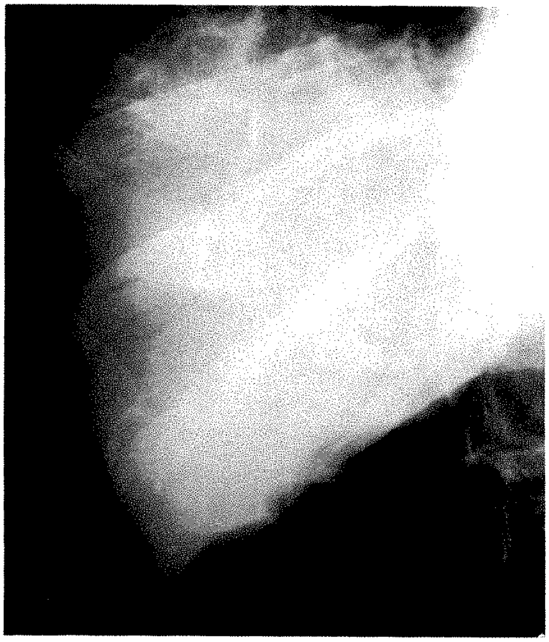

FIG. 2. Case I. Roentgenogram of the liver area on the following day demonstrates considerable decrease in the intravascular gas.

fringens. *E. coli* was cultured from the peritoneal cavity, which showed a mild generalized peritonitis. A postmortem blood culture was not obtained. In the pathologist's opinion, the cause of death was necrotizing colitis.

Comment. This case demonstrates diminution, and finally, disappearance of the intravascular gas on serial roentgenograms, a sequence of events which has been observed by others.[4] The inability to find intravascular gas at autopsy is in keeping with the roentgenographic findings.

CASE II. J. J., an 82 year old white woman, was admitted on March 11, 1963 complaining of epigastric pain, nausea and vomiting, increasing in severity over a 3 week period. She developed melena 3 days prior to hospitalization. On admission her blood pressure was 93/50 and pulse 122. The abdomen was distended with epigastric tenderness and guarding, and the bowel sounds were hypoactive. The stool was 3+ positive for occult blood.

Shortly after admission to the hospital, her blood pressure dropped to 60/0. Despite a temporary response to vasopressors, her condition progressively worsened, with increasing rigidity and distention of the abdomen, and coma. In spite of blood transfusions, oxygen, corticosteroids, and antibiotics, she died about 58 hours after admission.

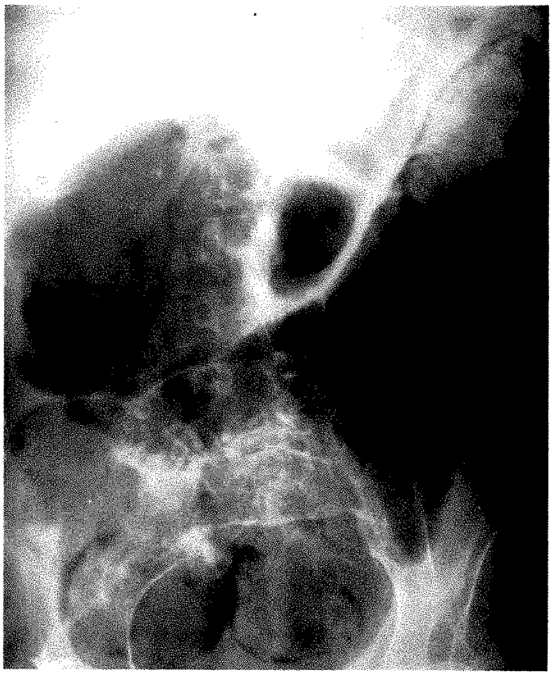

FIG. 3. Case I. Roentgenogram of the abdomen reveals complete disappearance of the portal vein gas. Intramural gas is now visible in the descending colon.

Roentgenographic Findings. Roentgenograms of the abdomen taken shortly after admission showed a moderately large amount of intestinal gas, predominantly in distended small bowel. Some gas was identified in the ascending and transverse colon, which was not dilated. These findings were those of a paralytic ileus. Several roentgenograms from an upper gastrointestinal series the following day (Fig. 4) revealed a linear, branching gas pattern in the liver region, interpreted as gas in the portal venous system.

Autopsy Findings. By careful dissection of the portal vein, numerous gas bubbles were noted within it, and these could be expressed from the vein when it was opened. No other intravascular gas was noted, and none was found in the biliary tract when the gallbladder was opened under water. The small bowel and proximal colon appeared necrotic, but no perforations were seen. Microscopically, the jejunum, ileum and right colon showed advanced

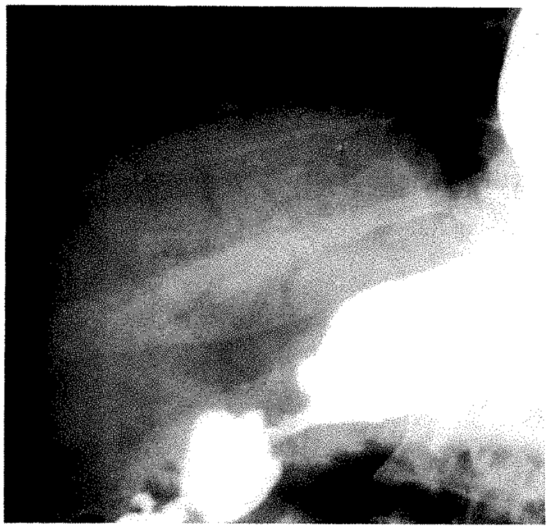

Fig. 4. Case II. Roentgenogram of the right upper abdomen shows gas within the portal veins of the liver.

necrotic and ischemic changes. Early ischemic and congestive changes were seen in the transverse and descending colon. Dissection of the mesenteric vessels failed to reveal any gross occlusive disease, but histologically there were advanced arteriosclerotic changes. There were recent and organized thrombi in the mesenteric veins. Postmortem blood culture revealed *Staphylococcus epidermidis*, while sections of the kidney and spleen grew *Clostridium perfringens*. *Clostridium perfringens* and *E. coli* were grown from the peritoneal fluid. The cause of death was considered to be bowel necrosis, pulmonary infarction, and bronchopneumonia.

Comment. Within a 24 hour period, this elderly woman developed gas in the portal venous system, and death followed 14 hours after its recognition. In view of the clinical history, and the type and distribution of the bowel lesions, the cause of the necrosis was due to ischemia, rather than necrotizing enterocolitis.

DISCUSSION

In 5 of the cases reported in the literature, portal venous gas has been associated with intestinal gangrene, either secondary to mesenteric artery thrombosis or necrotizing enterocolitis. Other underlying conditions were: erythroblastosis fetalis (3 cases), diabetes mellitus (2 cases), and 1 case each of imperforate anus, duodenal atresia, esophageal atresia, incarcerated obturator hernia, diarrhea, and small bowel necrosis (of unstated etiology). At postmortem examination, the majority of this group was found to have either necrosis of the bowel, or a compromised bowel wall, as indicated by congestion, ecchymosis, and/or distention.

The mechanism by which gas enters the venous system has not been established with certainty. One theory holds that gas escapes from the bowel lumen when under considerable pressure, and that the process is aided by areas of necrosis in the intestinal wall. This explanation does not account for some of the cases in which the integrity of the bowel wall is maintained. Numerous instances in which pronounced gut distention is present without the occurrence of intravascular gas also makes this theory less plausible.

We believe, as Wiot and Felson[4] do, that the gas is generated by gas-forming enteric organisms in the portal venous system. Gas-forming organisms found in both antemortem and postmortem blood cultures in several reported cases is certainly strong supportive evidence. Access of bacteria to the blood stream is facilitated by the intestinal distention and necrosis. The presence of pneumatosis cystoides intestinalis in at least 4 of the reported cases and in Case I above is an interesting associated finding.

Roentgenographically, the distinction between portal vein and biliary tract gas is based on the distribution of the linear branching radiolucent shadows. Gas in the portal venous system extends to the margin of the liver, whereas gas in the biliary tract remains centrally located in the area of the porta hepatis. It is thought that gas in the portal venous system is propelled by blood flow toward the periphery of the liver. In contrast to this, the caudal flow of bile acts against peripheral extension of biliary tract gas.

Portal vein gas appears to be a sign of imminent death. All the reported cases have succumbed within a short time, often a matter of hours. The longest reported survival after recognition was 8 days. It is doubtful whether the intravascular gas is directly responsible for the patient's death. More likely, it is an indication of overwhelming sepsis, frequently related to a gangrenous or seriously impaired bowel wall. Autopsy proof of portal vein gas is difficult to acquire, either because it may be overlooked, or it may be a transient phenomenon, as demonstrated in several cases. Follow-up roentgenograms are often difficult to obtain, either due to the patient's precarious condition, or rapid demise. Once the diagnosis has been established, energetic antibiotic and supportive therapy for the underlying condition should be carried out in an effort to turn the tide in the patient's favor. Being warned of the strong possibility of gangrenous bowel, the surgeon may plan his approach accordingly.

SUMMARY

Two cases of gas in the portal venous system are described. Although the mechanism by which this occurs has not been proven, the most likely possibility seems to be septicemia by a gas-producing microorganism entering the circulation through necrotic bowel. Intravascular location of the gas can be recognized by its peripheral extension in the liver area and thereby distinguished from biliary tract gas. Portal vein gas implies a grave prognosis. However, with increasing recognition of this entity and aggressive therapy, the chances of patient survival may improve.

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PROGRESSIVE POSTOPERATIVE PNEUMOPERITONEUM*

AIR ENTERING THROUGH DRAIN SITES

By D. JAMES HESLIN, M.D.,† *and* RONALD A. MALT, M.D.‡
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THE distinction between residual intraperitoneal air after abdominal surgery and pneumoperitoneum from a new disease entity during the postoperative period is sometimes a perplexing radiologic problem. Often the issue can be resolved by the demonstration of an increase in the amount of air over that which is usual, coupled with signs of a ruptured viscus, anastomotic leak, or infection with a gas-forming organism. Sometimes there are no clinical hints. In these situations, when the radiologist tests his diagnostic skill, he may also spare the patient needless surgery if he remembers that free air and progressive postoperative pneumoperitoneum sometimes may arise from as innocent a situation as air entering along a drainage tract.

REPORT OF A CASE

A 62 year old widow was admitted to the Massachusetts General Hospital for the eighth time complaining of watery diarrhea of 2 weeks' duration and of pain in the right side of her abdomen for 2 days. Previous admissions were for a transverse colostomy, sigmoid resection for diverticulitis, closure of colostomy, cholecystectomy, lysis of adhesions, and fever of undetermined origin with leukopenia.

She was a thin dehydrated woman with a temperature of 103.6°F. and a blood pressure of 115/60 mm. Hg. The abdomen was distended and tender, with high-pitched active bowel sounds. The hematocrit was 35 per cent and the white blood cell count 3,200 per cc. with 27 per cent polymorphonuclear leukocytes and 35 per cent band forms. Urinary specific gravity was 1.005 with 25 white blood cells per high power field in the sediment. The blood urea nitrogen was 20 mg. per 100 ml. Other laboratory findings were normal.

A ruptured appendix with a subhepatic abscess and a large inflammatory mass in the mesentery of the small bowel were found at laparotomy. One stuffed Penrose drain was placed in the immediate subhepatic area and one was placed with its tip at the foramen of Winslow and brought out through a right upper quadrant stab wound. Two other drains were placed in the right lower quadrant through a second stab wound.

Except for the presence of mild abdominal distention, the course after operation was uneventful. All drains were removed on the sixth postoperative day. On the following day her abdomen was distended. A roentgeogram of the abdomen on the eighth day (Fig. 1) showed large amounts of air in the peritoneal cavity including the lesser sac. The pneumoperitoneum was evacuated through a needle puncture in the epigastrium; clear serous fluid was also aspirated. The patient was afebrile and hungry but bothered with some mild diarrhea.

Abdominal distention persisted and pneumoperitoneum increased despite another paracentesis. Anteroposterior and upright roentgenograms of the abdomen showed intestinal obstruction and a massive pneumoperitoneum (Fig. 2 and 3). As the patient had manifested clinical signs of obstruction at this time, laparotomy was again carried out even though we suspected that the pneumoperitoneum could have been consequent on air entering through drain sites.

When the abdomen was opened, it was quite apparent that the stab wounds created a straight and freely open tract, wide at the outside and narrow inside, and there was absolutely no evidence of any fibrinous reaction or of any contraction in either one. It was easy to imagine a flutter-valve mechanism that would permit air to enter, but not leave, by these tracts. An extensive search of the entire bowel and pelvic organs revealed no site of other perforation. A mild degree of intestinal obstruction caused by a small abscess in the right lower quadrant was released.

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DISCUSSION

At least 2 cases of intraperitoneal air entering through drainage tracts have already been recorded.[1,2] In one, the amount of free air present in the left upper quadrant alongside a Penrose drain to the splenic bed was modest. In the second there was massive pneumoperitoneum. The source of this was a pair of rubber drains through the apex of the vagina into the peritoneum probably initiating a flutter-valve mechanism for the trapping of air.

The unique aspect of the case reported in this article is that the pneumoperitoneum actually became more pronounced after the drains were removed. In spite of the fact that the stab wounds were placed obliquely through the tissues, the thin abdominal wall and the absence of any

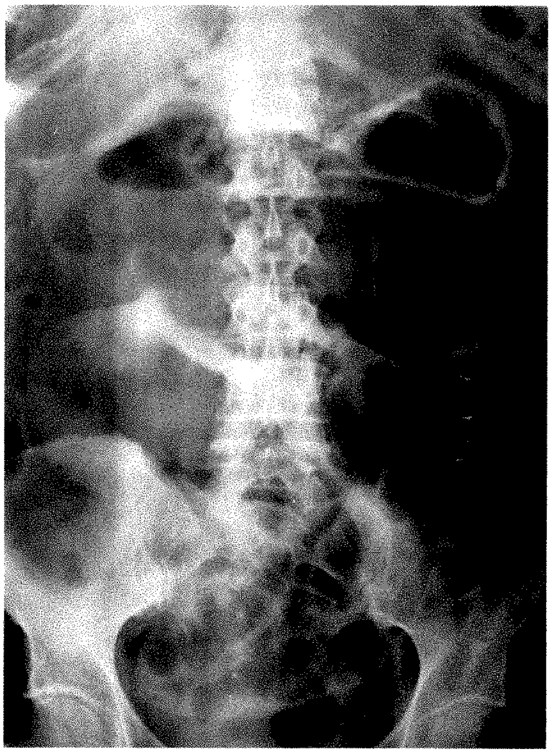

Fig. 2. Supine roentgenogram of the abdomen 1 day after Figure 1 showing progressive pneumoperitoneum and small bowel distention.

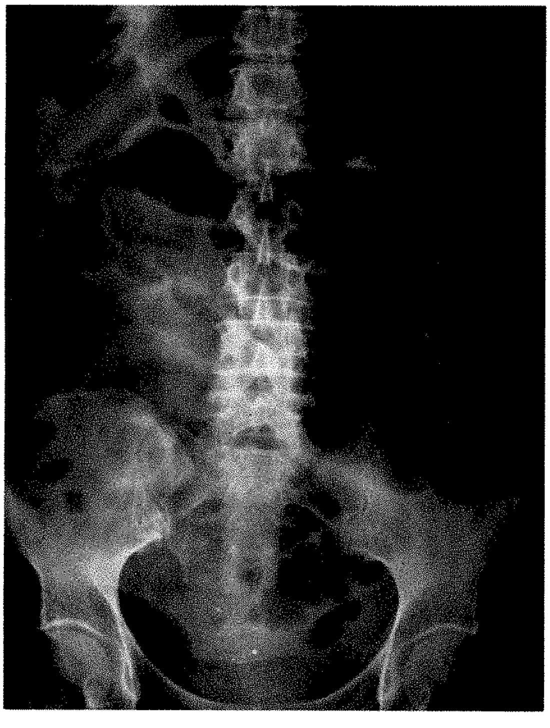

Fig. 1. Supine abdominal roentgenogram demonstrating free air throughout the abdomen and in the region of the lesser sac.

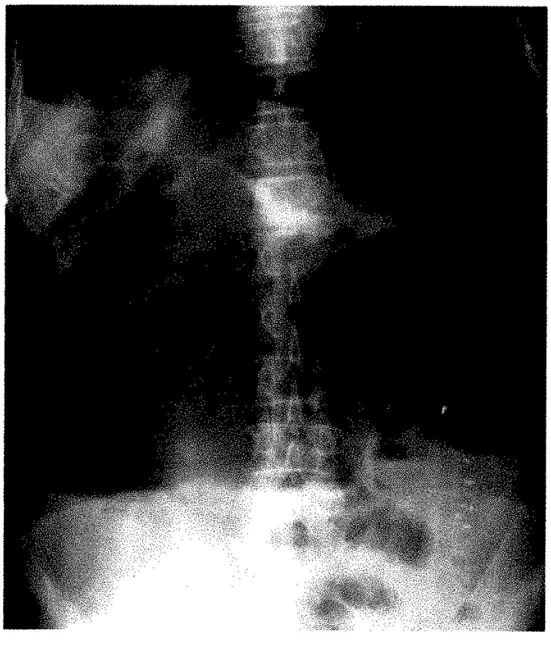

Fig. 3. Erect roentgenogram of the abdomen taken at the same time as Figure 2 showing distribution of air.

fibrinous response prevented the holes from sealing and led to the distention.

If drain sites are incriminated as a cause of pneumoperitoneum in a patient in whom indications for surgery do not otherwise exist, determinations of the PCO_2 of aspirated gas by conventional methods or of the H_2S or CH_4 content by gas chromatography[3] may help in deciding whether its origin is from without or within the abdomen.

SUMMARY

A case of pneumoperitoneum caused by room air entering the abdomen through drain tracts is reported.

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3. Karmen, A. Biological implications of gas chromatography. *Science*, 1963, *142*, 163–172

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Vol. 92, No. 5

NONFATAL INTRAVASATION OF BARIUM*

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IT HAS been estimated that several million barium enema examinations are performed yearly in the United States. This "routine" procedure, nevertheless, may be associated with complications of the utmost gravity, including perforation, water intoxication and intravasation. Perforation of the bowel is not uncommon and may involve the rectum due to overinflation of the Bardex bag, or may occur at the site of disease anywhere in the colon. Water intoxication is encountered, chiefly in individuals with megacolon.[1,3,9,11] Intravasation of barium is a rare complication, heretofore considered to offer a hopeless prognosis. In the American medical literature only 3 cases of intravasation of barium during enema study were found, with one survival. A fourth case, a survival, is added and is the basis of this report.

REPORT OF A CASE

A 65 year old Negro female was admitted to Meadowbrook Hospital on June 17, 1963, because of abdominal pain localized to the right upper quadrant. The pain had been present 2 days, radiated to her back and was associated with anorexia and vomiting. She appeared chronically ill and upon examination an irregularly outlined mass about the size of a grapefruit was palpated in the right upper abdomen. Its point of origin from kidney, liver or gallbladder was indeterminate. The remainder of the examination was negative. Her blood pressure was 160/100 mm. Hg. A diagnosis of hypertension had been made 1 year previously.

A roentgenogram of the chest was normal. An abdominal scout roentgenogram showed a large soft tissue mass in the right upper quadrant associated with a superimposed egg-shell calcification. An intravenous pyelogram resulted in poor excretion of contrast medium on the right. The appearance of this kidney was normal. The kidney was apparently separated from the mass. The gallbladder failed to visualize after attempted oral cholecystography. Digital and proctoscopic examinations were reported as normal and a barium enema study to establish the relationship of the mass to the colon was suggested.

The barium enema examination was performed utilizing the departmental routine. Preliminary roentgenograms confirmed the presence of the mass and calcification in the upper right abdomen. There was residual contrast material from the gallbladder study of the previous day (Fig. 1). A Bardex balloon catheter was inserted by a female assistant before the radiologist entered the room. On fluoroscopy the barium was seen to distend what was thought to be the rectal ampulla and a complete obstruction was encountered. The barium column failed to progress and after 2 to 3 minutes, a tangled mass of tubular structures of barium density was noted to radiate outward in all directions. The pattern assumed a "medusa-head" configuration. It was appreciated that the rectal venous circulation had been entered and the examination was immediately terminated. At no time did the patient volunteer any complaint of discomfort or pain. Immediate surgical and medical assistance was obtained and emergency prophylactic measures were instituted. Oxygen, penicillin, chloromycetin and corticosteroids were administered.

The patient was asymptomatic for about 20 minutes and vital signs were stable. She then began to develop a clinical picture of pulmonary embolization with tachypnea and a sense of suffocation. This episode lasted 20 minutes, whereupon she showed improvement. For the next 24 hours she experienced a mild cough and transient temperature elevation before reverting to her pre-examination status.

Roentgenograms showed the Bardex bag and a pocket of barium as well as the pelvic venous plexus. The pattern suggested a "medusa-head" (Fig. 2). A large roentgenogram clearly showed the left ovarian and renal vein as well as the pelvic venous plexus filled with barium (Fig. 3).

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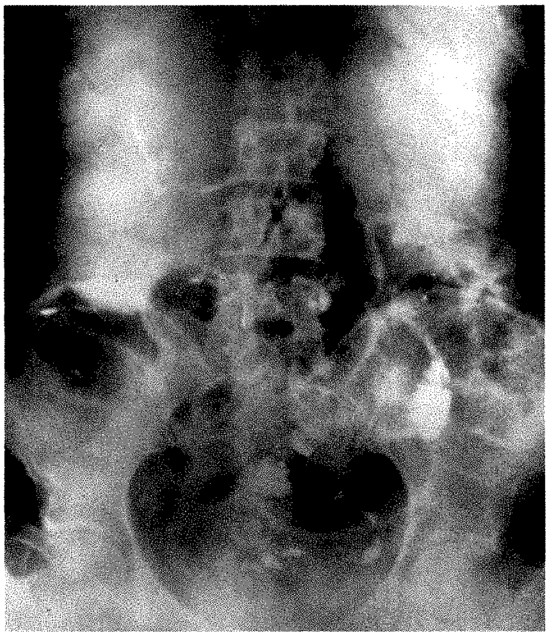

FIG. 1. Scout roentgenogram of abdomen showing mass in right upper quadrant displacing colon and crescentic calcification overlying mass. Residual contrast material from a previous cholecystography is seen in the colon.

The portal circulation was not outlined nor could barium be seen in the liver. A roentgenogram of the chest at this same time showed barium particles in both lungs (Fig. 4).

With stabilization of vital signs, a pelvic examination revealed barium in the vagina. A 3 inch laceration of the vaginal mucosa was found. The rectum was normal. It was postulated that the Bardex catheter had been inserted unwittingly into the vagina, and, upon distention, lacerated the vaginal wall and permitted barium to enter the venous system.

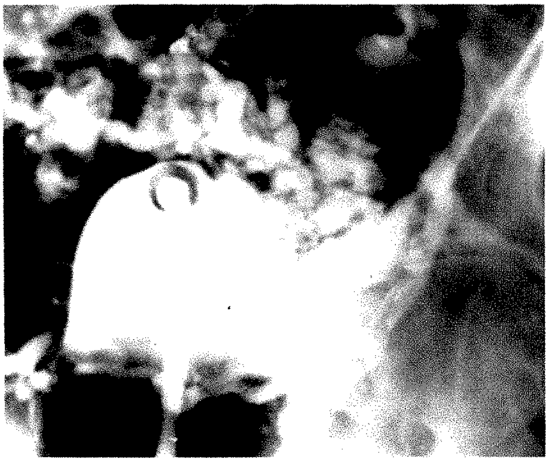

FIG. 2. Spot roentgenogram showing barium in pelvic venous plexus with "medusa-head" formation.

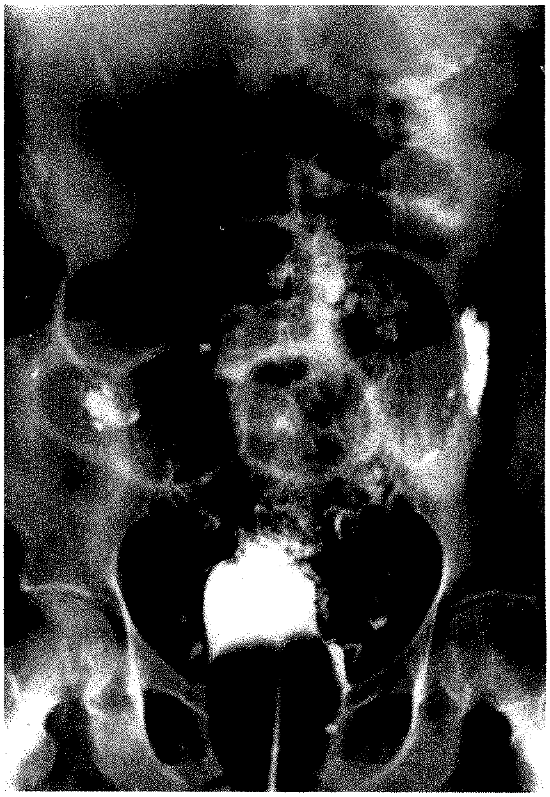

FIG. 3. Note barium in venous pelvic plexus. The descending left ovarian vein, left renal vein and renal venous plexus are demonstrated.

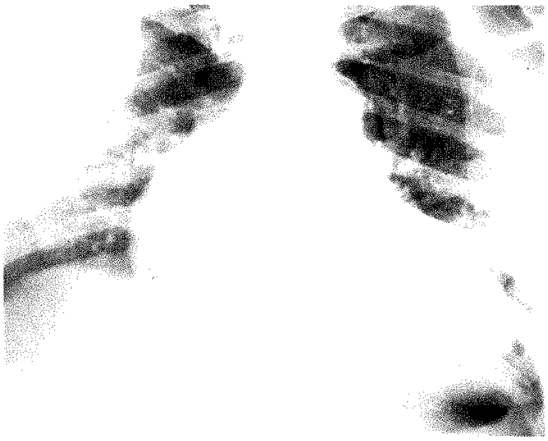

FIG. 4. Recumbent chest roentgenogram, made during clinical state of embolization, shows streaks of barium in right mid-lung and left upper lung.

Repeat roentgen studies within 24 hours failed to show barium in the lungs, liver or other organs. Three weeks later the barium enema examination was repeated with no difficulty and an intrinsically normal colon with downward displacement of the hepatic flexure was demonstrated. Exploratory laparotomy was performed and a mass adherent to the liver and occupying most of the right upper abdomen was found. The mass could not be resected. A biopsy was reported as "clear cell carcinoma, probably renal in origin."

The patient developed progressive congestive heart failure and pulmonary edema and she expired on the second postoperative day. Permission for autopsy was denied. Death was believed due to pulmonary edema following myocardial infarction.

REVIEW OF LITERATURE

The first case of barium intravasation was reported by Isaacs *et al.*[4] in 1950. They described a 25 year old female with ulcerative colitis who on postevacuation barium enema studies showed barium in the portal circulation. They postulated that the barium entered the veins as a result of mucosal weakening due to the ulcerative colitis. The patient was reported as being alive 2 years later. In 1952, Roman and his associates[7] reported a case of a 78 year old female, who died within 4 minutes following massive inadvertent intravenous intravasation of barium during a barium enema examination. Postmortem examination showed no potential point of origin for the embolization. The authors suggested entrance via a small venous fissure or varix. Rosenberg and Fine[8] in 1959 described a 73 year old female who died within 1 to 2 minutes after the start of a barium enema examination. Postmortem roentgenograms showed massive amounts of barium in the heart. No necropsy was done; the explanation offered was that rectal trauma due to the Bardex balloon provided entry for the barium.

DISCUSSION

The venous drainage of the pelvis is via vascular communication with both the systemic and portal systems. The rectum drains into the portal circulation through the superior hemorrhoidal vein and the inferior mesenteric vein, and into the systemic circulation through the inferior and middle hemorrhoidal veins. A free communication between portal and systemic venous systems is thus established through this hemorrhoidal plexus. The remaining pelvic organs, including the vagina, have extensive venous plexuses which drain into the hypogastric veins. In addition, the ovarian and spermatic veins drain into the renal vein on the left and directly into the inferior vena cava on the right. Barium thus can readily embolize to either portal or systemic circulations, and either liver or lung will act to filter out the emboli.

Intravasation of contrast material during hysterosalpingography and subsequent embolization to the lungs is well known. This is not considered an important or dangerous complication. Similar embolization of the contrast agent occurs during lymphangiography and results in no serious consequences. This may be due to the ability of the lungs to metabolize and excrete the two constituents of these contrast agents (iodine and a fat emulsion). No such metabolism of barium is known. However, it must be noted that the quantity of the embolized material is important. Massive amounts of any foreign substance, inert or organic, can find their way into the vascular system and cause serious symptoms, while smaller amounts are better tolerated.

The clinical picture of barium embolization is not characteristic. The severity of symptoms varies with the amount of material embolized and the size of the particles. With barium, the quantity intravasated would seem to be the decisive factor in affecting the outcome of the incident. The amount of barium demonstrated on postmortem roentgen studies in 2 of the reported cases seems to have been considerable. In Isaacs and co-workers' case of liver abscess following embolization, postevacuation roentgenograms showed only small amounts of barium in the vessels. In the case herein reported, prompt recognition of the accident resulted in a relatively small amount of barium entering the pelvic venous plexuses. This undoubtedly was responsible for the patient's survival. In the 2 fatal cases reported, shock and death ensued within 2 to 4 minutes, respectively. Our patient did not develop clinical signs or symptoms or embolization until 15 to 20 minutes after barium entered the venous system. This time lag permitted intensive medical measures to be instituted.

The accidental introduction of an enema catheter into the vagina is not a unique experience to most radiologists. It is generally a source of greater embarrassment than clinical importance. The patient usually protests vigorously and the error is quickly corrected. If the patient is too ill to react or unable to communicate, the examination may proceed with the radiologist under the impression that he is conducting an examination of the colon. This error may be discovered when the usual pattern of barium flow is not seen on the fluoroscopic screen. If an obstructing rectal lesion is suspected, the examiner may think he has encountered the obstruction and terminate the study with no harm being done. If a laceration of the vaginal mucosa is present prior to the examination or results from inflating the Bardex balloon, the pressure employed in the examination could easily force barium into the pelvic venous plexus. This pattern has a "medusa-head" appearance fluoroscopically. The procedure should immediately be terminated and medical measures should be instituted in anticipation of impending shock and pulmonary problems.

SUMMARY

1. A case of nonfatal barium intravasation via the vaginal venous plexus is reported.

2. The medical literature is reviewed.

3. The venous drainage of the pelvis is discussed.

4. The sudden appearance of a "medusa-head" barium pattern during fluoroscopic control of a barium enema study should alert the examiner to possible venous intravasation and calls for the immediate termination of the procedure.

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VOL. 92, NO. 5

MICROANGIOGRAPHIC STUDY OF GASTROINTESTINAL LESIONS*

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BY OPERATIVE arteriography benign and malignant lesions of the gastrointestinal tract are distinguishable.[8] The procedure is still considered experimental, and the methods and interpretation of results need further refinement. Deca-arteriography, the tenfold photographic enlargement of arteriograms, is one procedure that has improved the diagnostic accuracy and facilitated interpretation. A further step in magnification, however, was indicated: an evaluation of the advantages and limitations of microangiography. This method offers much further enlargement and detail. If histologic accuracy in diagnosis of gastrointestinal lesions is thereby achieved, methods to obtain similar magnification of vascular patterns *in vivo* must be developed. With this in mind microangiographic examination of resected specimens was undertaken. Efforts were made to (1) correlate vascular patterns with conventional histologic studies and (2) determine type and differentiation of vascular patterns of neoplasms. Could benign lesions be differentiated from malignant lesions; *e.g.*, a gastric ulcer from an ulcerated carcinoma?

METHODS

All specimens were removed surgically. As soon as feasible after excision, a small caliber polyethylene catheter was introduced into one of the main arteries supplying the lesion and tied in place. The medium used for injection was a mixture of micropaque* and thorotrast.† For micropaque suspension, 1 part of micropaque was mixed with 4 parts of water, which was then strained through gauze twice to eliminate clumps. Then 3 parts of this suspension were mixed with 1 part of thorotrast. This medium was injected intra-arterially under controlled pressure. The pressure varied from 150 to 200 mm. Hg but never exceeded this amount. Injection of the specimen was considered complete when the medium replaced the blood in the veins. This arterial and venous filling reproduced as nearly as possible the situation in *in vivo* arteriography.

Roentgenograms of the specimen were made before and after injection. The specimen was then opened and fixed flat in 10 per cent buffered formalin on a cork board. After a 24 hour fixation, roentgenograms were made of the opened specimen. Sections of the specimen, 1 cm. in thickness, were placed on edge for further roentgenograms.

From these various roentgenograms, areas were chosen for microangiographic examination and were marked on the films. Normal and abnormal sections were used whenever the quality of the injection so permitted. Tissue blocks of the chosen sites were cut and these in turn were sectioned with the Sartorius freezing microtome. A thickness of 500 micra was considered ideal. The remainder of the block was submitted for a routine section and stained with hematoxylin and eosin.

The 500 micra thick tissue sections were then blotted dry and placed between two pieces of Saran plastic. In a dark room, each section was tightly taped to the emulsion side of an Eastman Kodak spectro-

* Micropaque (Damany & Co., Ltd., Ware-Herts, England).
† Thorotrast (Testagar & Co., Detroit, Michigan).

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Part of the work was done at Washington University School of Medicine, St. Louis, Missouri.

TABLE I

LESIONS STUDIED

Stomach*		Colon†		Small Bowel	
Ulcer	8‡	Chronic Ulcerative Colitis	2	Carcinoid	2
Carcinoma	10	Cecal Ulcer	1		
Lymphoma	2	Carcinoma	14		
		Villous Tumor	1		
		Adenomatous Polyp	3		
		Metastatic Carcinoma	1		

* One specimen contained carcinoma and lymphoma.

† One specimen contained metastatic carcinoma and carcinoid of ileum and 2 an adenomatous polyp and a carcinoma.

‡ Five ulcers had been used entirely for histologic sections.

scopic plate, Type 649-D, measuring 1×3 inches. The plate was then placed in a small, light-proof cardboard box. A Picker Industrial X-ray unit with a beryllium window 0.5 mm. thick and a 1.5 mm. focal spot was used. The factors were 30 kv., 7 ma., at a 15 inch distance, with an average exposure of 10 minutes. The plates were processed for 5 minutes in x-ray developing solution and then placed in a fixer for 10 minutes. They were studied with a standard microscope. Diameters of vessels were measured with an eyepiece micrometer calibrated to the microscope.

RESULTS

Sixty-four specimens were injected; 40 were suitable for microangiographic studies (Table 1). Those considered usable had lesions and normal areas that were well injected although often some zones contained little, if any, contrast medium. The rejected specimens contained no areas with enough medium to adequately outline either normal or abnormal tissue. Even in the satisfactory specimens were areas in the normal mucosa that did not appear to have been well injected. Similar areas have been described by Nylander and Olerud.[12]

The vascular patterns of the mucosa, submucosa and muscularis of the stomach and of the small bowel and large bowel were basically similar (Fig. 1; 2; 7, *A* and *B;* and 8). The course of the serosal vessels roughly paralleled the muscularis with penetrating branches to the muscularis propria. In the muscularis the vessels formed a loose network, resembling a fish net. Vessels were seen to penetrate the muscularis to enter the submucosa. Through the muscularis, branches originated from these penetrating vessels. Occasionally, vessels that paralleled the major muscle layers were present. These served to set off the inner and outer muscular coats; the boundaries of the three muscle layers of the stomach were more difficult to determine. No difference was seen in the size or pattern of the vessels in the inner or outer muscular coats. The submucosal vessels were generally

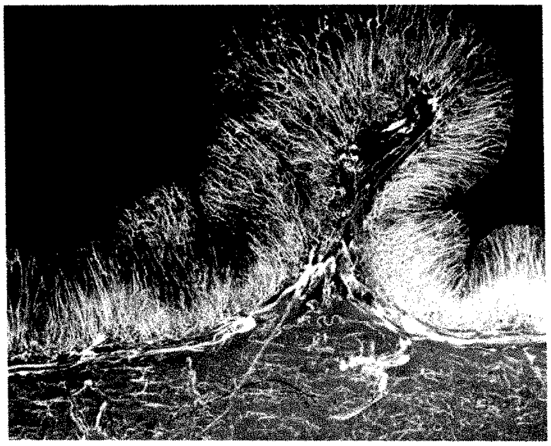


FIG. 1. Microangiogram of normal gastric wall. The relatively straight and numerous mucosal vessels are notable. The large submucosal vessels lie just beneath the mucosa and course horizontally in relation to the mucosa. Next the lacy network of the muscularis is seen. Large piercing vessels are present (×7. B.U.C.M. #13507-A).

larger than those of the muscularis and tended to be horizontal to the mucosal vessels. Branches from the submucosal vessels penetrated and spread into the muscularis mucosae and then into the mucosa. The muscularis mucosae was difficult to identify by a vascular pattern; it appeared on microangiograms to be in the zone of angulation of vessels into the mucosa from the submucosal vessels.

Vascular patterns of the mucosa at various sites in the gastrointestinal tract were remarkably similar. The major mucosal vessels were relatively straight with small branches at varied intervals. Near the surface the vessels formed a complex branching network. The pattern was much like that described by Jacobson and Noer.[6]

In some sections of small bowel, the villous pattern was distinct and aided identification; large bowel and stomach were often indistinguishable when studied as unknowns. Occasionally, in sections from the gastric antrum or pylorus, clusters of Brunner's glands were identified by a network of fine vessels in the submucosa. The vessels were seen to surround the gland units. The vessels of Brunner's gland averaged 11.7 micra in diameter compared to 15.5 micra for those of the mucosa. A difference between arteries and veins could not be seen on the microangiogram. Both were filled, as was readily evident on the hematoxylin and eosin stained sections.

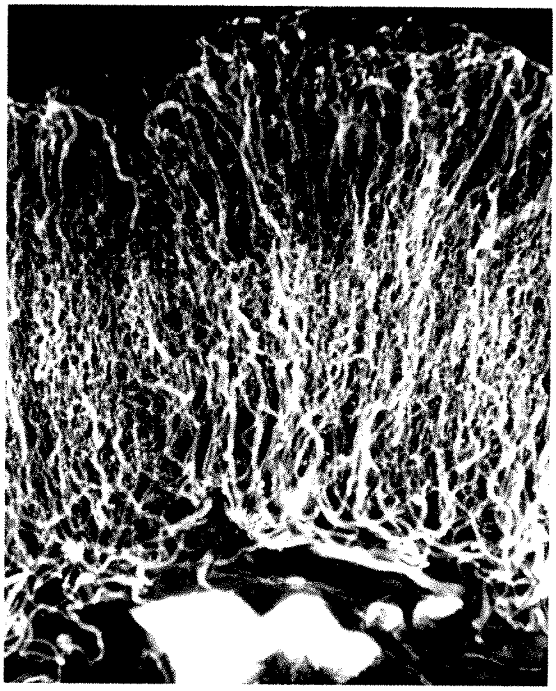


Fig. 2. Higher magnification of normal stomach mucosa. The intricate network of vessels can be seen in the inner two-thirds of the mucosa. Larger vessels may be traced from base to surface. The caliber of the larger vessels changes very little from bottom to top (×75. W.U. Ill. #62-4341).

To establish a baseline for comparison with the abnormal, the diameters of the injected vessels in the layers of normal bowel and stomach were measured. The measurements did not include many of the larger vessels, which were often incompletely filled. All of the measurements were of apparently completely filled vessels. Another source of error in obtaining exact diameter measurements was the cutting of vessels at different levels in the preparation for microangiography. The vessels for measurement were taken at random for representative diameters. Mucosal vessels were measured at different points of their lengths. Little change in diameter was noted from base to termination.

In the gastrointestinal tract, the diameters of the measured vessels were similar regardless of site (Table II). In the colon, a variation in the diameter of mucosal vessels was noted at different sites. The mucosal vessels of the rectosigmoid were smaller (9.7 micra) than those in the cecum or transverse colon (12.2 and 13.5 micra, respectively). The submucosal and muscularis vessels of the cecum were likewise larger than those in other colonic sites. The diameter of the submucosal vessels averaged 70.8 micra. Muscularis vessels in the cecum measured 17.6 micra. In the sigmoid, the measurements were 31.8 and 12.5 micra, respectively.

Generally, the vascular pattern of all the lesions studied varied definitely from the normal. The deviations were often spectacular, especially in neoplasms, but less so in the non-neoplastic diseases. The vascu-

TABLE II

DIAMETERS OF NORMAL VESSELS IN MICRA

Organ	No. of Measurements	Diameter Range	Average Diameter
Stomach			
Mucosa	312	5–75	15.5
Submucosa	151	5–420	73.1
Muscularis	196	5–150	17.7
Small Bowel			
Mucosa	33	3.7–18.7	12.8
Submucosa	15	12.5–150	66.9
Muscularis	20	7.5–25	15.9
Large Bowel			
Mucosa	209	5–34.2	11.8
Submucosa	136	6.2–374	55.2
Muscularis	167	5–60	14.1

lar pattern of the inflammatory lesions usually presented many fairly straight vessels, whereas in the neoplasms the pattern was chaotic except in well differentiated neoplasms where it resembled the normal.

The measurements of lesion vessels are tabulated in Table III. The considerably greater diameter than that of normal vessels, excepting the submucosal, is apparent. Thus, in addition to the pattern changes, the diameter of the vessels of gastrointestinal lesions is altered.

STOMACH

Gastric Ulcer. Microangiograms of ulcers in this study were obtained during acute exacerbations of chronic ulcers. Roentgenograms of the injected whole specimens and of the sections demonstrated an aggregation of vessels in the area of ulcer which imparted a rather diffuse haziness (Fig. 3, *A*, *B* and *C*). The vessels appeared to be radiating toward the ulcer bed (Fig. 3*D*). These findings were similar to those reported by others.[5,7] The tissue was much more vascular than normal. The vessels tended to be straight and of uniform caliber. As seen in Tables II and III, the diameters of normal vessels differed from those of an ulcer. One ulcer bed contained vessels that were less well oriented to the surface and were tortuous. Tortuosity was less, however, than that in carcinoma and the vessels were of more uniform diameter. Granulation tissue was exceedingly vascular. The vessels were numerous and large, and their course was sinuous. Microangiographically, granulation tissue was difficult to distinguish from carcinoma. Inflammatory lesions composed of large quantities of granulation tissue could present difficulties in the interpretation of *in vivo* arteriograms.

Mucosal proliferation at the edge of ulcers contained larger vessels, slightly tortuous and shorter than the normal mucosal vessel. The vessels of the submucosa and muscularis were not much altered in these areas. Distant from the immediate ulcer zone, the gastric vasculature appeared normal.

Gastric Carcinoma. In general, carcinoma caused a definite alteration of the vascular pattern that corresponded roughly with the histologic differentiation (Fig. 5, *A*, *B* and *C*). The poorly differentiated and diffusely infiltrating carcinomas presented vascular patterns that bore no resemblance to the normal.[14] Well differentiated carcinomas, on the other hand, maintained a vasculature in which certain normal pat-

TABLE III

DIAMETERS OF LESION VESSELS IN MICRA

Disease	No. of Measurements	Diameter Range	Average Diameter
Gastric Ulcer	31	7.2–275	46.6
Gastric Carcinoma	159	5.0–500	42.7
Gastric Lymphoma	16	6.3–45	24.5
Small Bowel Carcinoid	30	3.7–145	31.3
Chronic Ulcerative Colitis—Mucosa	69	7.5–55.8	22.2
Cecal Ulcer	20	6.3–70.0	33.0
Large Bowel Carcinoma	263	5.0–400	52.5
Large Bowel Polyps	39	7.2–46.8	25.4
Villous Tumor	11	6.2–30	15.3
Metastatic Carcinoma to Cecum	10	10–210	59.0

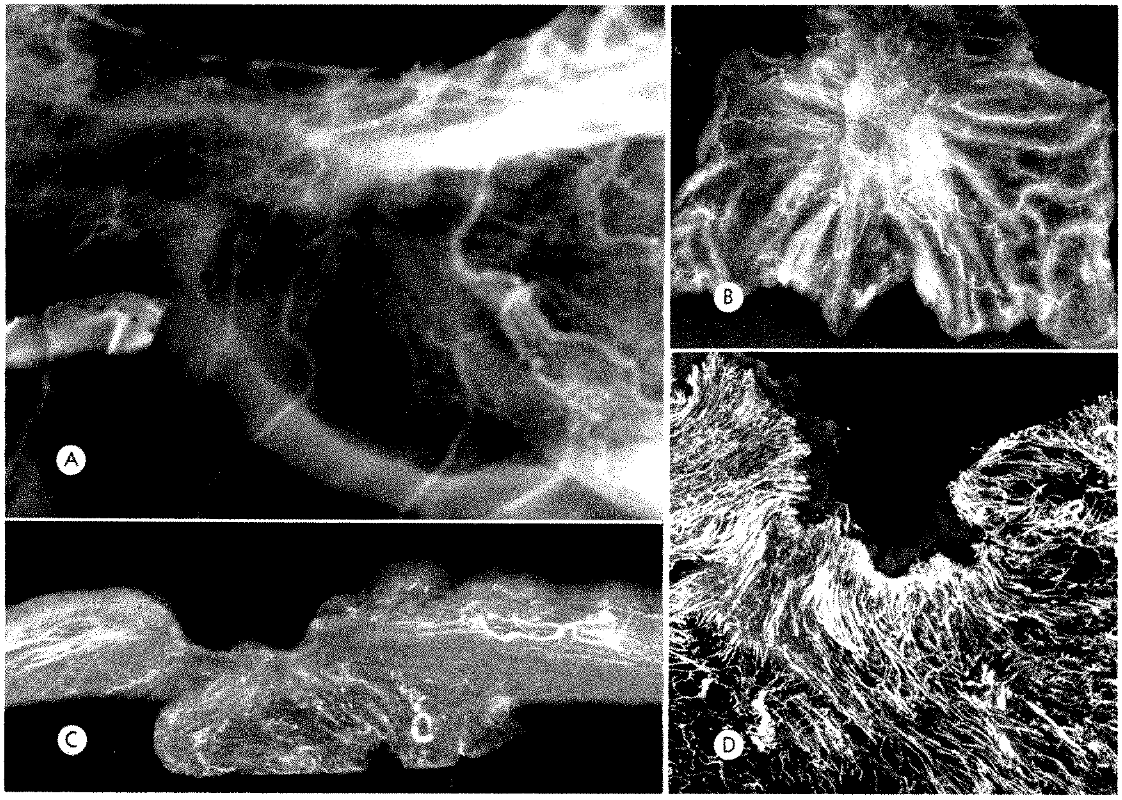
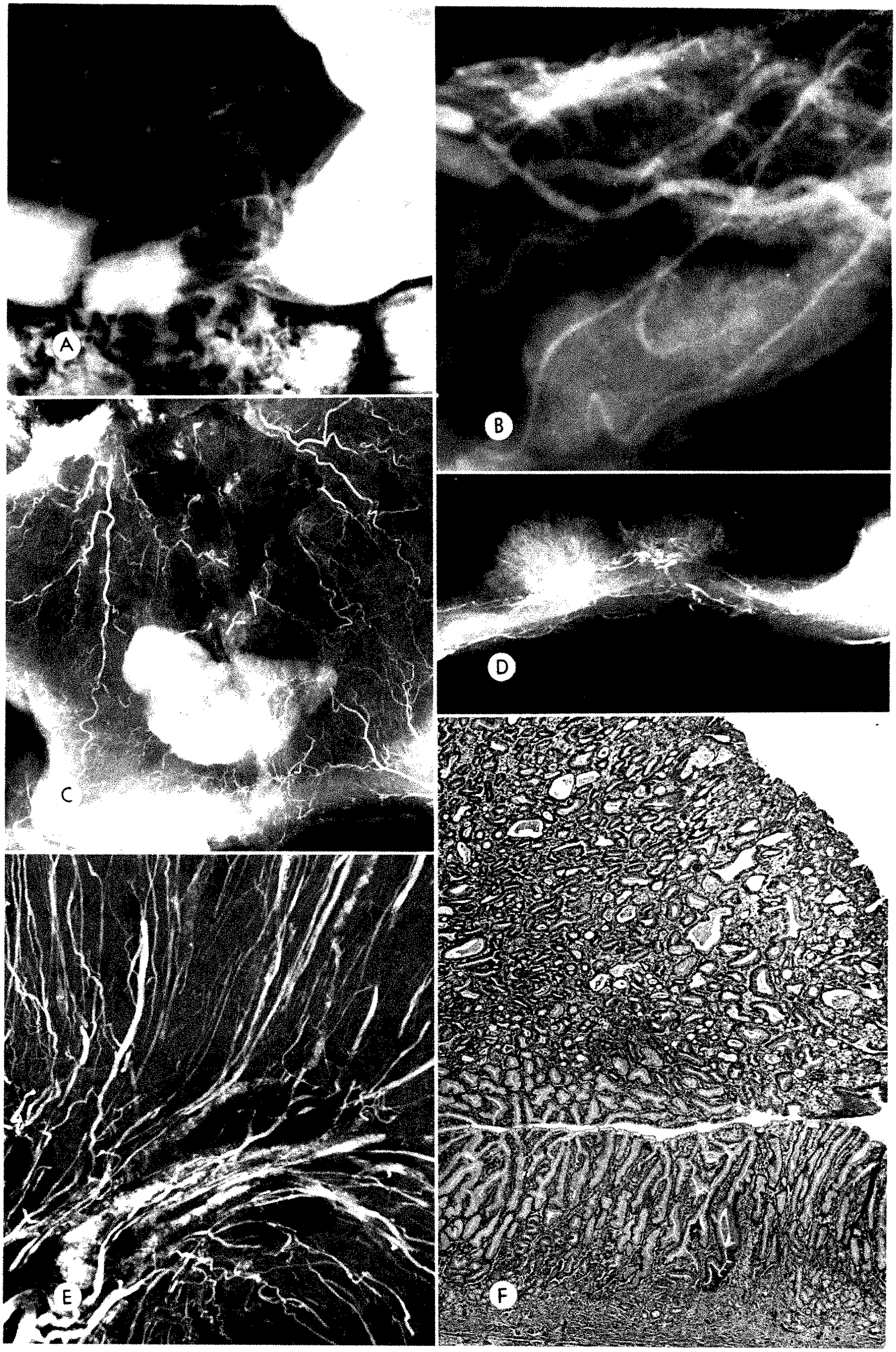


FIG. 3. (*A*) *In vivo* arteriogram of a gastric ulcer (W.U. Ill. #61-8150). (*B*) An injected, opened, and fixed stomach with an ulcer. The ulcer is a circular, lighter stained area in the most vascular part of the specimen. The vessels give the impression of streaming toward the ulcer. The large vessels are in the submucosa (B.U.C.M. #13507). (*C*) A 1 cm. thick section of the ulcer and adjacent gastric wall of the specimen seen in *A*. There is streaming of vessels toward the ulcer. Thickened vascular perigastric fat is attached. The mucosa is represented as a brush-border, seen best to the left of the ulcer (×10. B.U.C.M. #13507). (*D*) Microangiogram of the gastric ulcer seen in *A* and *B*. The bed is extremely vascular when compared to normal. The vessels seem to radiate toward the ulcer. Histologically, the ulcer base was dense scar tissue. To the right of the ulcer, distorted but nearly normal mucosa approaches the ulcer edge. The erupting vascular mass in the left half of the ulcer is a thrombosed artery. The gray material covering the ulcer is exudate (×12. W.U. Ill. #62-4334).

terns could be recognized (Fig. 4, *A*, *B*, *C*, *D* and *E*).

The vessels of a carcinoma were usually of greater diameter than normal, of variable caliber, and devious in their course. In areas of necrosis the vessels were sparse or absent. When tumor widely replaced layers of the stomach, the normal vascular pattern was lost. With focal involvement, however, this normal pattern was maintained but the neoplastic clusters had the pattern previously described. When carcinoma cells infiltrated mucosa but the glands persisted, the mucosal vascular pattern remained recognizable. One small ulcerated carcinoma contained vessels of two patterns: the usual carcinomatous configuration and the ulcer pattern. The latter was manifested by vessels appearing to spread toward the ulcerated surface just as seen in the benign ulcer (Fig. 6, *A* and *B*). In view of the significance of infiltrating and "pushing" margins of tumors,[9] of interest was one of the carcinomas with pushing margins in which vessels conformed to this pattern. The vessels at the periphery of the tumor tended to bend with the advancing edge.


A
B
C
D
E
F

ulcerative colitis with a neoplasm by *in vivo* arteriography is not likely. In fact, arteriography may develop to the point where it would be precise in the detection of carcinomas in ulcerative colitis. The vascular patterns of carcinomas are probably distinct enough to differentiate carcinoma from surrounding chronic ulcerative colitis.

Adenomatous Polyp and Villous Tumor. The vascular patterns of the three polyps and the villous tumor resembled the normal mucosa (Fig. 12 and 13). For the most part, the vessels were long and straight without much change in caliber from base to the mucosal surface. In foci of atypical glandular proliferation within a polyp, the regular arrangement of the vessels had become disorganized. No groups of vessels bore a semblance to carcinoma. The one villous tumor was not well injected. In those areas seen well enough for study, no difference could be seen in the vasculature that set apart a villous adenoma from adenomatous polyps.

Colonic Carcinoma. Both in the intact injected specimen and in the 1 cm. thick sections, the carcinomas were distinguishable from the normal by their extreme vascularity (Fig. 12). No features differed from those described for the stomach carcinomas.

Microangiographically, the colonic carcinomas were similar to gastric carcinomas. The vessels were equally as chaotic in pattern and as varied in caliber. Vessels at the surface of ulcerated carcinomas resembled those seen in granulation tissue. Some actually opened onto the surface. Areas of necrosis in tumor were devoid of vessels; extravasated injected contrast material was not seen (Fig. 14, *A* and *B*; and 15, *A* and *B*).

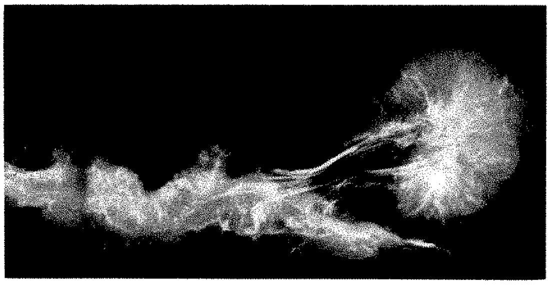


FIG. 12. A 1 cm. thick section of a carcinoma and a polyp of the sigmoid colon. Even with the low magnification, the rather orderly pattern of the vessels of the polyp contrasts with the irregular one of the carcinoma (×5. B.U.C.M. #13507).

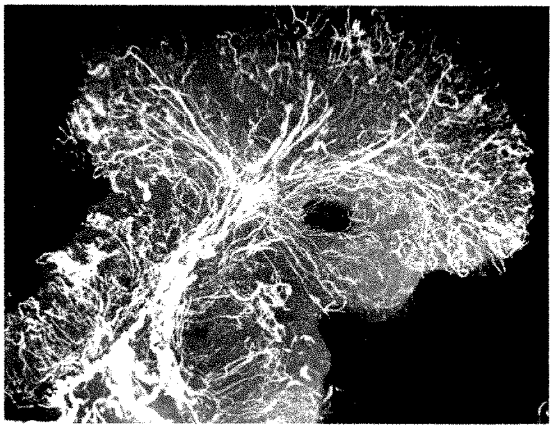


FIG. 13. Microangiogram of an adenomatous polyp with focal atypical changes. The stalk is set off by the large vessels. In general, the pattern simulates normal mucosa. The nests of tortuous vessels represent atypical glandular foci (×9. B.U.C.M. #13507A).

Near areas of transition between tumor and normal mucosa, the vascular pattern of the mucosa resembled the normal. In these zones carcinomatous and non-neoplastic glands intermingled.

The poorly differentiated carcinomas could not be distinguished from the well differentiated.

Metastatic Carcinoma to Colon. The one case studied was a metastatic renal cell carcinoma. Of all the lesions, this was the most vascular (Fig. 16, *A*, *B* and *C*). The pattern was unlike that of the primary carcinomas. Instead of the longer winding vessels of carcinoma, this lesion was composed of small vascular units. The small tumor components left a nonopaque area imparting a sponge-like pattern to the tumor. This correlated well with the histologic sections of the mass and the known rich vascularity of renal cell carcinoma.

DISCUSSION

Many organs and tissues have been studied microangiographically,[1-4,10,15,16] and

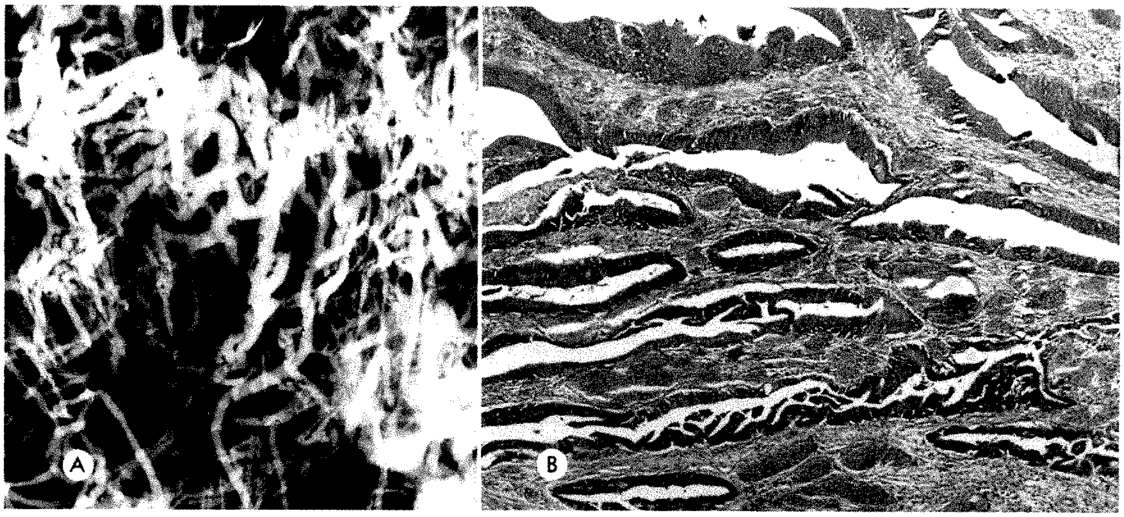


FIG. 14. (*A*) Microangiogram of a fairly well differentiated adenocarcinoma of the splenic flexure. The tangle of abnormal vessels can be seen. Individual vessels demonstrate kinks and irregular dilatations (×29. B.U.C.M. #13507A). (*B*) Photomicrograph of the same lesions as in *A*. The granular material in the blood vessels is the contrast medium (×35. B.U.C.M. #13507A).

many different techniques have been used.[1–3,7,8,10,11,13] The vasculature of the normal gastrointestinal tract in man and animals has been studied rather carefully. In our material the vascular patterns of the gastrointestinal tract proved to be similar to those previously described. That many factors will alter the normal vasculature and probably that of neoplastic and non-neoplastic diseases is understandable. What effect such factors as anesthesia, drugs complementing anesthetics, other drugs, or nutrition may have on vessels in lesions is not entirely known. These may or may not influence interpretation of arteriograms.

Measurements of normal and abnormal vessels were obtained within the limitations noted. They serve, however, as a

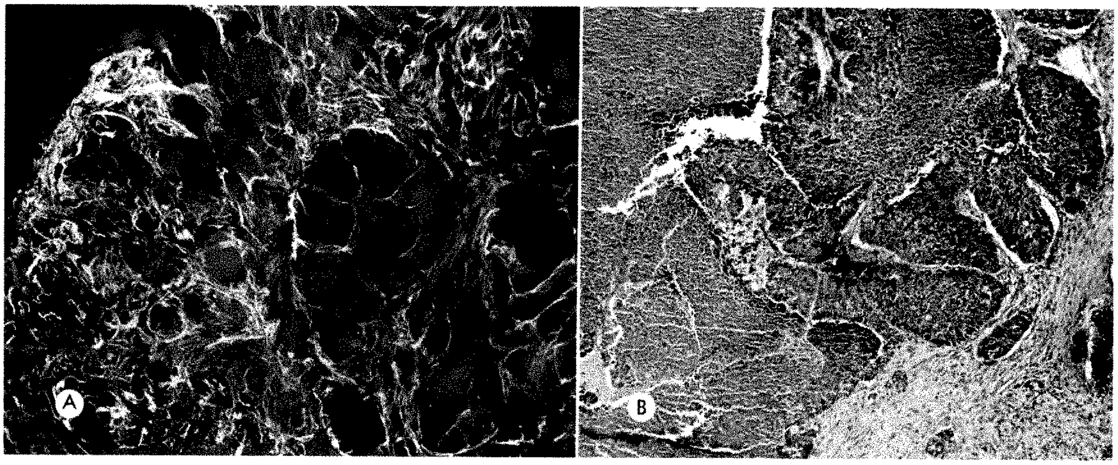


FIG. 15. (*A*) Microangiogram of an ulcerated, poorly differentiated adenocarcinoma of the hepatic flexure. Tumor is present in nearly all portions of this illustration. The circumscribed gray-black areas in the tumor represent foci of necrosis. These are sparsely vascular (×12. W.U. Ill. #62-4530). (*B*) Histologic section from the same block as in *A*. The lobules of necrotic tumor are seen with viable carcinoma at the periphery (×85. W.U. Ill. #62-4534).

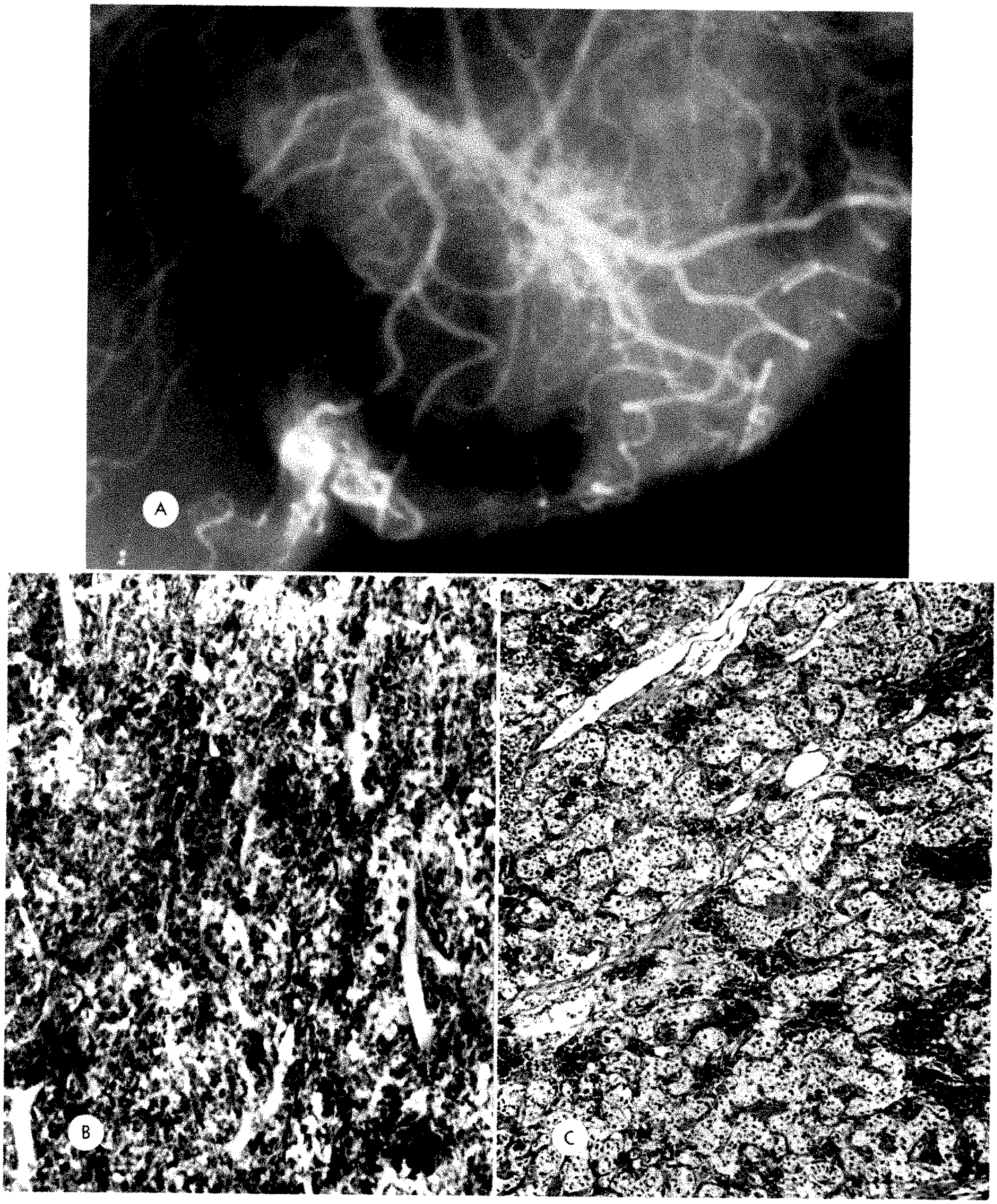


Fig. 16. (*A*) *In vivo* arteriogram of metastatic renal-cell carcinoma to the cecum (W.U. Ill. #63-2330). (*B*) Microangiogram of the lesion seen in *A*. This was the most vascular tumor that was studied (×35. W.U. Ill. #62-4663). (*C*) Histologic section from the same block as in *B*. Characteristic renal-cell carcinoma is seen. The myriad small blood vessels contain the micropaque-thorotrast mixture (×85. W.U. Ill. #62-4669).

graphic means of comparing the lesion vessels with the normal. Of interest is the difference, although not great, of the caliber of vessels in different parts of the gastrointestinal tract (Table II). The significance is not apparent but may reflect to some degree the various physiologic functions of the parts.

In general, the findings obtained with microangiography correlate well with the arteriographic and histologic observations. They dramatically demonstrate the vascularity of tissues and lesions, a vascularity known to exist but not readily appreciated. With deca-arteriograms, individual vessels may be studied and details of the vascular patterns of tumors become evident. Microangiography brings these into sharper focus. The vascular pattern of cancers is with few exceptions diagnostic. No benign lesion that we studied, with the possible exception of granulation tissue, resembled a malignant one. Even with granulation tissue, when the surroundings are considered, a mistake would not be made. This may, however, not prove true with conventional arteriography as the hypervascularity and tortuosity of the vessels could be interpreted as neoplastic.

Differentiation of the carcinomas did not seem to alter the vascular pattern of malignant growth to any great degree. The only notable exception was the well differentiated polypoid carcinoma of the stomach (Fig. 4*E*). In this lesion the vessels were long and straight, similar to the pattern of adenomatous polyps. From this one example, benign polypoid lesions of the gastrointestinal tract might be difficult to segregate from the malignant on the basis of vascular studies alone.

Vascularity of tumors and its patterns may influence the choice of treatment, especially in regard to radiation therapy and chemotherapy. Since all of the malignant tumors studied were hypervascular, chemotherapeutic agents would be assumed to reach all except the necrotic zones of a tumor. Although hypervascularity does not necessarily relate to oxygen content, a possible relationship between irradiation and tumor vascularity and pattern might be considered.

SUMMARY

Microangiographic studies were made of 40 gastrointestinal lesions. Coincidentally, normal stomach and gut wall were examined in a similar manner.

The normal vascular patterns are described. Measurements of the diameters of normal vessels were compared with the abnormal. Although the vascular pattern of different parts of the gastrointestinal tract is similar, the caliber of the vessels varied, the gastric having the largest diameter, the colonic the smallest.

The vascular pattern of malignant lesions is strikingly altered from the normal. The vessels are increased in number and are chaotic in arrangement. Benign lesions are hypervascular but the vessels are not in much disarray as in the malignant lesions.

On the basis of vascular patterns, *in vivo* arteriography and deca-arteriography should offer a valuable diagnostic aid in distinguishing many lesions.

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PHYSICAL FACTORS IN CINEFLUOROGRAPHY*

EXPOSURE METER AND PHANTOM MATERIALS

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IN PLANNING a series of studies of the physical characteristics of cinefluorographic apparatus,[2,3] we found that there were two technical problems which had to be considered at the outset.

The first problem was the design of a suitable exposure meter. The dose rates encountered with cinefluorographic apparatus range from about 10 r/min. with peak exposure rates as high as 100 r/min. during air calibration measurements, to about 1 mr/min. when measurements are made of the exposure rate at the input phosphor of an image intensifier tube. Thus the exposure meter must have a dynamic range of at least 10^5. An accuracy of better than 2 per cent should be maintained through this range.

Water is accepted universally as the best phantom material with which to simulate absorption of x rays in patients. In making measurements of the characteristics of cinefluorographic apparatus as a function of simulated patient thickness, however, a thin solid absorbing material must be used so that the absorber thickness can be changed by small increments. Accordingly, we investigated absorption characteristics of pressdwood to determine its suitability as a substitute for water.

In this paper the results of our consideration of these two problems are summarized.

I. EXPOSURE METER

We found that an exposure meter with characteristics that considerably exceeded the minimum specifications could be assembled from commercially available components. A block diagram of our final design is shown in Figure 1; the performance characteristics are shown in Table 1. This exposure meter combines the advantages of a null system[1,4] and a direct reading system.[5] Some increase in sensitivity and range has been gained by the use of a vibrating capacitor electrometer, at the sacrifice of portability.

The Philips ionization chamber most suitable for diagnostic exposure measurements is the Type 37482 which has a volume of about 1 cc. This chamber was calibrated in our exposure meter by comparison with a Victoreen 25 r ionization chamber that had in turn been calibrated at the National Bureau of Standards. The results of the calibration are shown in Figure 2, where it is seen that the response of the chamber is essentially uniform from 0.5 mm. Al HVT (half-value thickness) to 3 mm. Al HVT. The response of the chamber gradually rises until it is up by 10 per cent at 9 mm. Al HVT. The ion recombination loss of the instrument was studied by measuring the apparent exposure rate as a function of polarizing potential applied to the ionization chamber. A half-wave rectified x-ray generator was used as a radiation source. It was found that the ion recombination loss is less than 1 per cent with a polarizing potential of 180 volts when the average exposure rate is less than 1,000 r/min. This would correspond to an average exposure rate of 2,000 r/min. with a full-wave rectified apparatus or a peak exposure rate of about 4,000 r/min.

Neither the accuracy of the electrometer nor the cable capacitance affects the accuracy of exposure measurements when the exposure meter is operated in the null mode. It is often convenient, however, to use the exposure meter as a direct reading device. Under these conditions, a correction

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Table I

DOSIMETER CHARACTERISTICS

Reading Mode	Component	Mid-Range Sensitivity	Time Constant	Exposure Range			
				Minimum Limiting Condition		Maximum Limiting Condition	
Null	500 μμF C	1.59 r/v	—	1.6 mr	(1)	2.55 r	(2)
Null	0.0535 μF C	177 r/v	—	177 mr	(1)	283 r	(2)
Direct	500 μμF C	3.10 r/v	—	3.1 mr	(1)	4.96 r	(2)
Direct	0.0535 μF C	179 r/v	—	179 mr	(1)	286 r	(2)
Direct	73.5 MΩ R	2,710 $\frac{\text{r/min.}}{\text{v}}$	0.03 sec.	≈3.5 r/min.	(3)	~2,000 r/min.	(4)
Direct	7,350 MΩ R	28.0 $\frac{\text{r/min.}}{\text{v}}$	3 sec.	140 mr/min.	(3)	840 r/min.	(5)

Limiting Conditions:
1. 2 per cent precision in reading potentiometer at null (1 mv.).
2. Maximum voltage from potentiometer (1.6 v.).
3. 2 per cent precision in direct reading 10 mv. electrometer scale (at 5 mv.).
4. Less than 1 per cent ion recombination loss, full wave rectified.
5. Maximum electrometer voltage (30 v.).

must be made for the cable capacitance and the linearity and the accuracy of the electrometer become important. The linearity and accuracy of the electrometer were tested by applying known voltages, determined by a potentiometer with a rated accuracy of 0.05 per cent. It was found that all scales were linear, and that the upper $\frac{8}{10}$ of all scales were accurate, to better than 1 per cent. In the direct reading mode of operation, cable capacity reduces the sensitivity of the instrument by a factor of 1.945±0.004 when the 500 μμF capacitor is used; with the 0.0535 μF capacitor, the cable capacitance reduces the sensitivity of the instrument by 1.1 per cent.

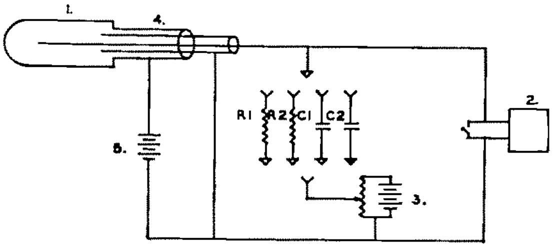

Fig. 1. Schematic diagram of exposure meter. C1: 500 μμF, Air; C2: 0.0535 μF, Mylar. R1: 73.5 MΩ; R2: 7,350 MΩ. (1) Chamber: Philips, 37482, 1 cc. volume. (2) Electrometer: Cary (Applied Physics Laboratory) Model 31. (3) Potentiometer: Leeds and Northrup #8687. (4) Cable: Philips F-37485/0 with matching connectors. All other cables Amphenol 621-715 or RG 59U. All other connectors Amphenol BNC. (5) Polarizing batteries: 180 v.

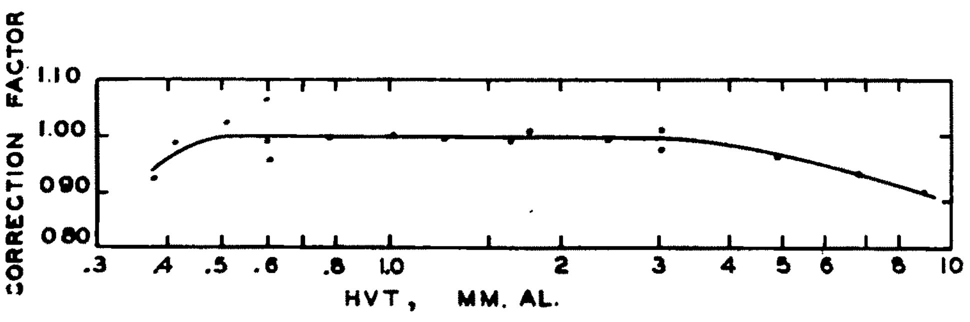

Fig. 2. Energy response of Philips 37482 chamber in exposure meter. See Table 1 for calibration factors.

Possible stem leakage effects were investigated by irradiating varying lengths of the stem at HVT 2 mm. Cu. It was found that, within the 1 per cent precision of the experiment, the stem leakage is negligible when as much as 21 cm. of the ionization chamber stem is irradiated. However, care must be taken not to irradiate the connector between the ionization chamber and the cable, as this introduces spurious effects.

The zero drift of the electrometer corresponds to an exposure rate of less than 0.002 mr/min.

This exposure meter has operated satisfactorily for 2 years at the time of writing. It should also be noted that it is adaptable for use with any of the Philips ionization chambers. For example, with the Type 37486/10 high energy chamber, it has been used for the measurement of transmission dose with a 2 mev. Van de Graaff unit and for the measurement of exposure rates from 1 mg. sources of radium and iridium 192. The sensitivity of the instrument can be increased appreciably, at the sacrifice of a larger chamber volume, by utilizing the 30 cc. Type 37488/10 ionization chamber.

II. PHANTOM MATERIALS

Materials used to simulate the interaction of diagnostic x-ray beams in patients must: (1) be equivalent to water, the accepted standard absorber, and (2) be available in thin sheets so that incremental changes in thickness can conveniently be made. Of solid materials, Mix D is probably most nearly equivalent to water but it is difficult to obtain in thin sheets, is expensive, and is brittle. Pressdwood, which has often been used as the absorber in investigations in diagnostic radiology, does not have the disadvantages of Mix D, but it is variable in composition and in manufacture. It should be legitimate, however, to use pressdwood as an absorber if the relationship between transmission of x rays through it and through water is demonstrated for the particular lot used.

For this comparison, measurements were made of the exposure dose transmitted through variable thickness of water and a particular lot of pressdwood. An intermediate therapy apparatus was used as the x-ray source. The quality of radiation was varied from HVT 3 mm. Al to HVT 6 mm. Al. Transmitted exposure was measured with the exposure meter described in the first part of this paper. A second exposure meter was used to monitor the incident exposure. Care was taken to establish geometry for measurement so that the contribution of scattered radiation from the absorbers was negligible. The thickness of the pressdwood was expressed in gm./cm.^{2} by correcting for the density of this particular lot of pressdwood, 0.97. The results of the comparison are shown in Table II and Figure 3.

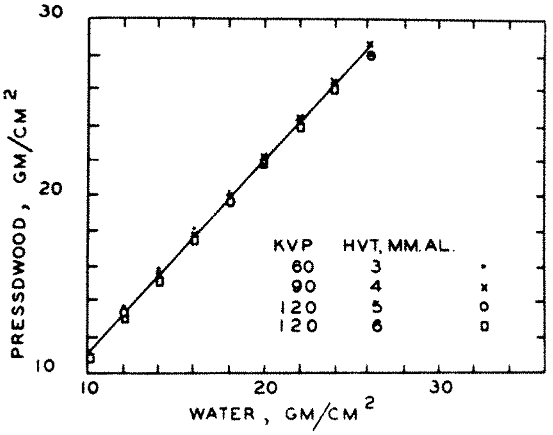

FIG. 3. The thickness of pressdwood (vertical axis) required to produce the same attenuation of an x-ray beam as the thickness of water specified on the horizontal axis. Data for the various x-ray beams as designated.

It can be seen that there is a linear relationship between pressdwood thickness and water thickness for the same absorption of the x-ray beam and that this relationship is almost independent of beam energy over the range considered. An analysis of the data, however, showed that the ratio of the pressdwood thickness to water thickness, in gm./cm.^{2}, for equal transmission of exposure varies from 1.125 at HVT 3 mm. Al to 1.085 at HVT 6 mm. Al. Apparently, the effective atomic number of the pressdwood that was tested is slightly greater than that of water. This observation im-

Table II

COMPARISON OF MATERIAL THICKNESS FOR EQUAL TRANSMISSION OF X RAYS

kvp.	60	90	120	120
HVT, mm. Al	3	4	5	6
Water Thickness (gm./cm.^{2})	Pressdwood Thickness (gm./cm.^{2})			
10	11.3	10.9	10.9	10.7
12	13.7	13.3	13.3	13.1
14	15.9	15.6	15.4	15.3
16	18.2	17.8	17.6	17.5
18	20.2	20.1	19.9	19.8
20	22.2	22.2	21.8	21.8
22	24.2	24.4	24.1	23.9
24		26.6	26.2	26.2
26		28.8	28.1	28.1

plies further that the ratio should decrease, at a given HVT, as absorber thickness increases. This effect was shown to be just significant ($P=0.025$) at the lowest HVT but could not be demonstrated for the higher energy beams. If a mean value for the ratio, 1.105 on a gm./cm.^{2} basis or 1.14 on a cm. basis is used, however, the error in calculating equivalent water thickness introduced by assuming energy independence will be less than 2 per cent, over the energy range considered. This approximation is probably sufficiently accurate for practical purposes.

It should be noted that this result may not apply to any other lot of pressdwood.

SUMMARY

A versatile exposure meter with wide dynamic range and excellent precision that is suitable for measurement of exposure in diagnostic radiology and has been assembled from commercially available components is described.

An investigation of pressdwood as a suitable absorber to simulate patient absorption in the diagnostic x-ray region showed that for a particular lot of pressdwood, the relationship between pressdwood thickness and water thickness was linear and nearly independent of energy in the region from HVT 3 mm. Al to HVT 6 mm. Al.

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PHYSICAL FACTORS IN CINEFLUOROGRAPHY*

RELATIVE PATIENT DOSE FOR 35 MM. AND 16 MM. CAMERA SYSTEMS

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MOST cinefluorographic machines can be equipped with either a 35 mm. or 16 mm. motion picture camera and lens system. While there are many aspects of the two camera sizes that can be compared, the purpose of this paper is to evaluate only one: the relative patient dose required for equal motion picture film exposure with the two camera systems. Measurements were made with a cinefluorographic unit that is equipped with a variable x-ray tube potential system to maintain a desired luminance of the output phosphor of the image intensifier tube,† but the data also can be used to predict the behavior of a cinefluorographic machine in which the variable x-ray tube current method of exposure control is used.

METHOD

The luminance of the output phosphor of an image intensifier tube as a function of the dose at the entrance surface of a patient may be calculated if three characteristics of the cinefluorographic apparatus are known: (1) the air exposure rate calibration, (2) the transmission of radiation through water equivalent absorbers of various thickness to the input phosphor of the image intensifier tube, and (3) the conversion factor of the image intensifier tube (*i.e.*, output phosphor luminance/input phosphor exposure rate). The luminance of the output phosphor required to produce equal photographic exposure of the motion picture film can be determined experimentally for a given film type and for the two camera systems. From these data, the patient dose that is necessary for equal exposure of the film can be found for the two camera systems.

AIR EXPOSURE RATE

The air exposure rate characteristics of a typical cinefluorographic machine are shown in Figure 1. These data were obtained with the equipment and methods that have been discussed previously.[4] The manufacturer's calibration was accepted for the potential of the x-ray tube, but measured half-value thicknesses over the range of potential are given in the figure inset. With a precision of ±0.1 mm., the half-value thickness is independent of x-ray tube current.[4] The x-ray tube current stations are designated by the manufacturer's arbitrary letter nomenclature because of the well-known difficulty in relating x-ray tube current as indicated on a meter to exposure dose rate with fluoroscopes.[7]

FRACTIONAL TRANSMISSION

The transmission data (Fig. 2) are expressed in terms of the fractional exposure rate at the front face of the image intensifier tube referred to the exposure rate in air at table top. The data were obtained by removing the image intensifier from the carriage of the tower of the fluoroscope and placing the ionization chamber at the same level as the front face of the image intensifier tube. The lowest projection of the

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† The G. E. Fluoricon cinefluorographic unit used for the measurements reported in this paper has been described previously.[4] The unit has a target-to-table top distance of 18.7 inches and operates with 2 mm. Al added filter.

* From the Department of Radiology, Hospital of the University of Pennsylvania.

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carriage was maintained 1 cm. from the top surface of the absorber. Since the distance between the lowest projection of the carriage and the front face of the image intensifier tube was 17.7 cm., the total distance between the top surface of the absorber and the ionization chamber was 18.7 cm. The radiation was filtered by the same components as in clinical operation: added absorber to simulate a patient, the phototimer, a 6:1 grid with 60 lines/inch, and the bakelite protective cover of the image intensifier tube. Although, for convenience, pressdwood sheets were used as the added absorber, the thickness was converted to its equivalent water thickness by methods that have been discussed previously.[5] Since the radiation beam was composed of both primary and scattered radiation, the cap/no cap ratio method[2,3] was used to estimate the half-value thickness of the emergent beam shown in the inset of Figure 2. Appropriate corrections were made for the energy dependence of the ionization chamber. The field size was adjusted just to circumscribe the image intensifier tube input phosphor for each absorber thickness; thus, the field size was always 9×9 inches at the measuring distance.

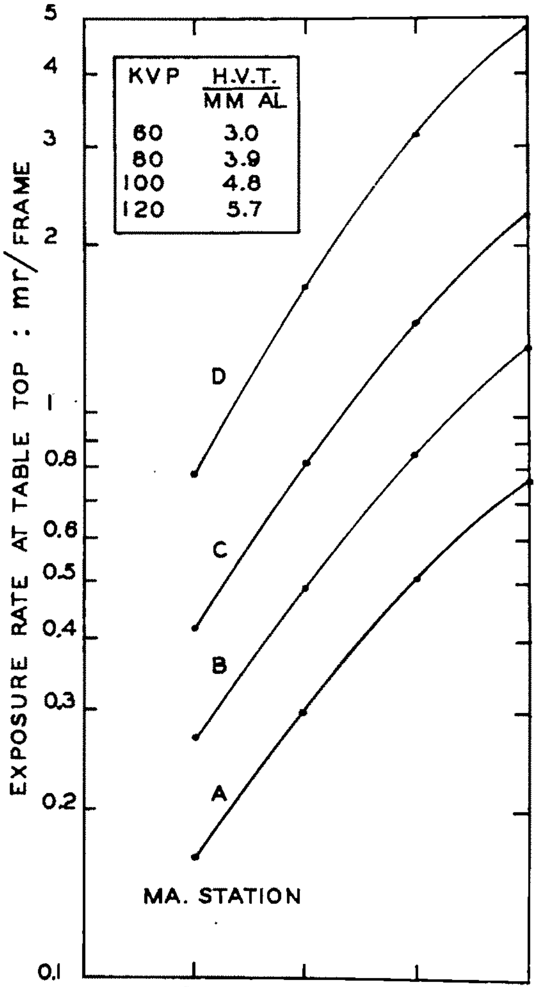

Fig. 1. Calibration of exposure dose rate in air at level of table top for cinefluorographic unit. The four available milliampere stations are designated by the letters.

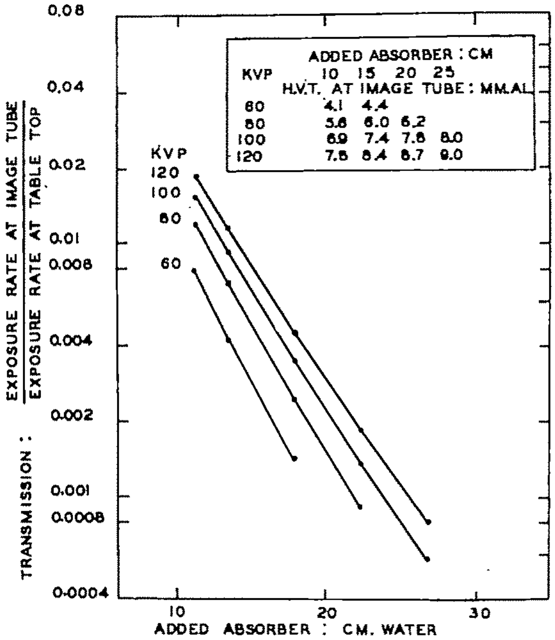

Fig. 2. Fractional transmission data through water equivalent added absorber for radiation produced by cinefluorographic unit. See text for conditions of measurement.

CONVERSION FACTOR OF THE IMAGE INTENSIFIER TUBE

The optical system of the image intensifier was removed so that direct measurement of the luminance of the output phosphor could be made with a Spectra SB 1½° Brightness Spot meter. Luminance data

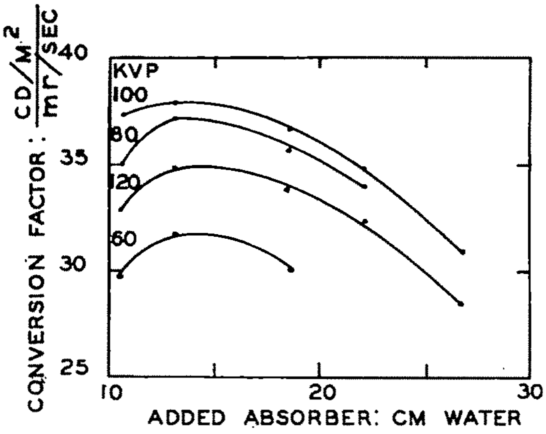

Fig. 3. Conversion factor of image intensifier tube as a function of the added absorber that simulates patient thickness. The conversion factor is the ratio of the luminance of output phosphor (candela/meter^2) to the exposure rate at the input phosphor (mr/sec.).

were obtained over a wide range of x-ray tube potential and absorber thickness with the apparatus operated as a fluoroscope. The exposure rate at the input surface of the image intensifier tube* was calculated from air exposure rate curves, similar to those of Figure 1, and the transmission data of Figure 2.

The data obtained for the ratio of the luminance of the output phosphor to the exposure dose rate at the input surface, that is, the conversion factor of the image intensifier tube, are shown in Figure 3. The results are presented in this form to exhibit the strong dependence of the conversion factor on absorber thickness. A cross plot of conversion factor as a function of potential, for any fixed absorber thickness, gives a curve with a broad maximum at about 95 kvp., as reported and discussed by Henny and Becker.[6] Luminance is related linearly to exposure rate, for any fixed tube potential, over the range in which measurements were made (0.1 to 10 foot-lamberts; 1 to 100 mr/min.).

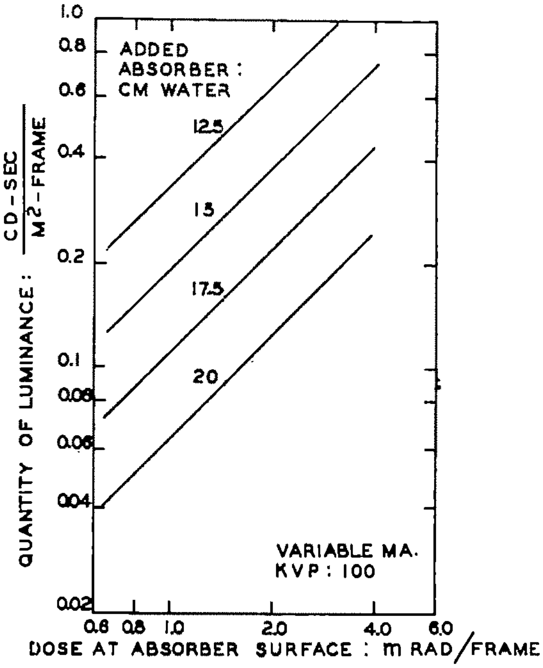

Fig. 4. Typical results of calculation of the quantity of luminance at the output phosphor of the image intensifier tube as a function of the dose rate at the surface of the absorber that simulates the patient. These data are for cinefluorographic operation with the variable x-ray tube current method of exposure control of the motion picture film and with the x-ray tube potential fixed at 100 kvp.

OUTPUT PHOSPHOR LUMINANCE AS A FUNCTION OF PATIENT DOSE

With the data that have been presented in Figures 1–3, the quantity of luminance at the output phosphor can be calculated as a function of the absorbed dose at the front surface of the absorber for a wide range of conditions.† Typical results are shown in Figure 4, for the case of variable x-ray tube current at a fixed x-ray potential, and in Figure 5, for variable potential with fixed current.

The relation between quantity of luminance at the output phosphor and absorbed x-ray energy for variable x-ray current, Figure 4, is linear, as expected. For a fixed tube potential, the fractional transmission of the x-rays through the absorber and the

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* The image intensifier was a CFTH 9 inch diameter tube.

† Backscatter factors were taken from the *British Journal of Radiology*, Supplement 10. The rad/r conversion factor was taken from the 1959 ICRU recommendations, NBS Handbook 78. Luminance measurements were made with a meter calibrated in foot-lamberts and with the cinefluorograph operating at 30 frames/second. These data were converted to the preferred system, candela/meter,^{2} and quantity of luminance was obtained by multiplying luminance by the number of seconds per frame.

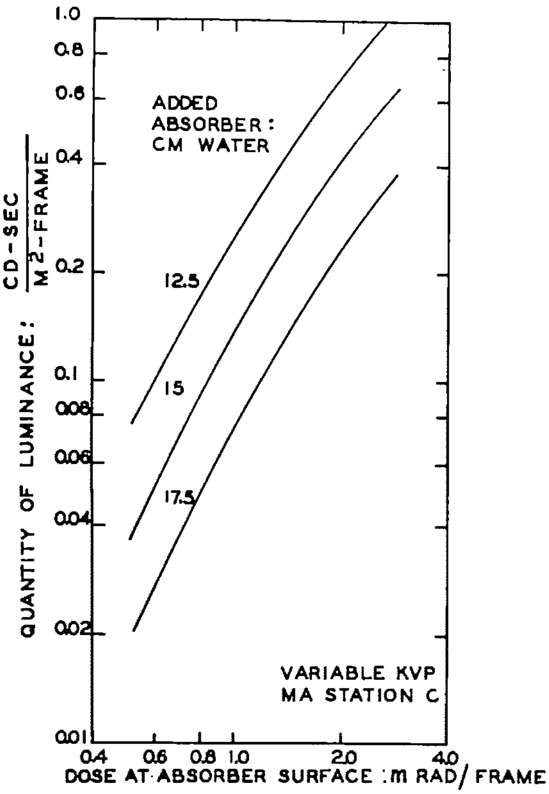

Fig. 5. Results similar to those of Figure 4, except calculations are for the variable x-ray tube potential method of motion picture film exposure control. X-ray tube current fixed at milliampere station C in this example.

conversion factor of the image intensifier tube are constant. The light produced by the image intensifier tube, accordingly, should be proportional to the dose rate produced by the x-ray tube and this, in turn, should be proportional to the x-ray tube current.

The functional relation for the curves in Figure 5, is more complex. To produce more light in operation with constant x-ray tube current, the dose rate of the machine is increased by increasing the x-ray tube potential. At the same time, the penetrating power of the radiation increases, and, in the potential range from 60 to about 100 kvp., the conversion factor of the image intensifier increases. Thus, the emitted light energy would be expected to be a power function of the absorbed x-ray dose. At potentials above 100 kvp., the slope of the curve decreases because of the decrease in the image intensifier tube conversion factor.

OUTPUT PHOSPHOR LUMINANCE FOR EQUAL FILM EXPOSURE WITH 16 MM. AND 35 MM. CAMERA SYSTEMS

With the cinefluorographic unit in the automatic mode of operation at 30 frames per second, the 35 mm. camera system, and an absorber equivalent to 13.3 cm. of water, a series of film strips was exposed with various settings of the exposure level control ("cine brightness factor"). The x-ray tube current and the potential that the automatic exposure control assumed for each setting of the exposure level control were noted. The camera system was then changed to the 16 mm. size and the experiment was repeated.* Plus-X Negative film was used, as a representative medium speed, medium detail film. Strips of the 16 mm. and the 35 mm. film were also simultaneously exposed to light under a transmission step wedge as a control on film emulsion and processing. All film strips were processed simultaneously for 4 minutes at 80° F. in an automatic processing unit using Liquid X-ray Developer. Comparison of the densities of the strips exposed under the step wedge showed that the emulsion characteristics of the two film sizes were identical, within experimental error. Film response curves were plotted from the net film density and exposure level setting data obtained from the strips exposed in the cinefluorographic unit. From these curves, the setting of the exposure level control which gave equal density for the two camera types could be determined. Finally, reference to the x-ray tube potential and current, previously recorded, allowed calculation of the quantity of luminance at the output phosphor, through the use of the data of Figures 1–3, as previously discussed. This sequence of measurements was repeated after the 35 mm. camera system was once again installed in place of the 16 mm. system. The exposure rate of the x-ray machine was calibrated before and after each experimental run, and the coefficient of variation was found to be slightly greater than 2 per cent. The coefficient of variation of the precision with which the exposure control system adjusted to constant output phosphor luminance in automatic operation was found to be about 4 per cent by measuring the relative illuminance at the focal plane of the motion picture camera over a wide range of operating conditions and absorber thickness.

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* In this apparatus, standard General Electric cinefluorographic cameras were used in both cases with a Bausch and Lomb 90 mm., f/1.0 objective lens. The 16 mm. lens was an Angenieux 25 mm., f/0.95, T-stop 1.2. The 35 mm. lens was a Bausch and Lomb Baltar, 75 mm., f/2.3, T-stop 2.4. The two camera lenses were calibrated in the T-stop system so that their true relative speed could be determined. The T-number is defined as the f-number of an ideal lens having 100 per cent transmittance and a circular aperture which would give the same central image illuminance as the actual lens at the specified stop opening.[9]

It was found that the 35 mm. camera system required a quantity of luminance of 0.4±0.02 cd-sec./m.^{2}-frame at the output phosphor of the image intensifier tube to produce a net film density of 0.82±0.05, whereas the 16 mm. camera system required a quantity of luminance of 0.089 ±0.005 cd-sec./m.^{2}-frame to produce the same net film density.

RELATIVE PATIENT DOSE FOR 16 MM. AND 35 MM. CAMERA SYSTEMS

With the results of the previous paragraph and a set of curves such as Figures 4 and 5, the relative patient dose necessary to obtain equal exposure of the film in the two camera systems can easily be determined. The results are given in Table I for the cinefluorographic unit with the variable current method of exposure control, and in Table II for operation with the variable potential method. The accuracy of these values is believed to be no greater than about ±8 per cent, because of the indirect method by which they were calculated. A direct experimental test of the method of calculation was performed, however, at one milliampere station. The x-ray tube current and potential assumed for automatic variable potential operation were noted as a function of absorber thickness with settings of the exposure level control appropriate for the two camera systems. The dose to the absorber could be calculated directly from the air exposure rate calibration of the machine, Figure 1, with corrections for

Table I

PATIENT DOSE RATIO FOR EQUAL FILM EXPOSURE: VARIABLE X-RAY TUBE CURRENT EXPOSURE CONTROL

$\frac{35 \text{ mm.}}{16 \text{ mm.}}$ Camera System

Kvp.	Absorber Thickness (cm.)	Dose Ratio
80	10.5	4.5
	12.5	4.5
	15	4.4
100	15	4.5
	17.5	4.5
	20	4.4
120	17.5	4.6
	20	4.4
	22.5	4.5

Table II

PATIENT DOSE RATIO FOR EQUAL FILM EXPOSURE: VARIABLE X-RAY TUBE POTENTIAL EXPOSURE CONTROL

$\frac{35 \text{ mm.}}{16 \text{ mm.}}$ Camera System

Ma.	Absorber Thickness (cm.)	Dose Ratio
A	10.5	2.6
	12.5	2.9
B	10.5	2.4
	12.5	2.6
	15	2.9
C	12.5	2.4
	15	2.4
	17.5	2.7
D	15	2.5
	17.5	2.5
	20	2.7

backscatter and the rad/r conversion factor. These measurements agreed within 5 per cent with the calculated values.

DISCUSSION

Comparison of 16 mm. and 35 mm. camera systems may be made for a number of parameters. The cost of 35 mm. film, for example, is higher, as are the processing and viewing facilities. On the other hand, the better resolving power of a 35 mm. system can be demonstrated with high contrast test objects.[8] Many believe, however, that this difference is not significant in clinical practice with presently available image intensifiers.[1]

The two systems may also be compared on the basis of the patient dose required to produce acceptable motion picture film exposure. The difference in patient dose is principally due to differences in the lens systems. Physical design problems and space limitations dictate that a lens for 35 mm. film be appreciably slower than a lens for 16 mm. film in cinefluorography. Thus to produce equal film exposure for the 35 mm. system, the image intensifier must produce more light and the x-ray dose to the patient must be higher.

For a cinefluorographic unit which employs the variable x-ray tube current method for exposure control, the light produced is proportional to the current, as has been noted in the discussion of Figure 4, so the 35 mm./16 mm. patient dose ratio should be determined by the speeds of the two camera lenses. This argument is in fair agreement with the results presented in Table I. The speed advantage of the lens used for 16 mm. film is $(2.4/1.2)^2=4.0$.

The patient dose for a cinefluorographic unit which uses the variable potential method of exposure control is also greater for a 35 mm. than for a 16 mm. camera system, but the difference is not as great as in the case of variable current exposure control because at a fixed x-ray tube current the system with the 35 mm. camera always operates the x-ray tube at a higher potential and, therefore, more efficiently than the system with the 16 mm. camera. Thus, the light produced by the image intensifier tube increases more rapidly with increase in patient dose, as has been discussed in connection with Figure 5. At lower operating potentials, the 35 mm./16 mm. patient dose ratio is about 2.5 (Table II). As the patient thickness increases, the system becomes less efficient because of the fall-off of the image intensifier tube conversion factor but the 35 mm./16 mm. patient dose ratio stays below 3. With this method of exposure control, however, the radiographic contrast will decrease as patient thickness increases. An evaluation of this effect is beyond the scope of this paper and the assessment of its clinical significance would be complicated by consideration of the diagnostic information desired in various types of examinations. Generally, it can be said that the variation in contrast is not believed to be detrimental by the diagnostic staff of this department.

SUMMARY

One of the factors by which 35 mm. camera systems for cinefluorography can be compared to 16 mm. camera systems is the patient dose required to produce equal motion picture film exposures. Measurements have been made of the characteristics of a cinefluorographic machine from which the ratio of patient dose has been calculated for 35 mm. as compared to 16 mm. camera systems with either variable x-ray tube potential or variable x-ray tube current methods of motion picture film exposure control. For the variable current method of exposure control, the 35 mm./16 mm. patient dose ratio was found to be 4.5, in fair agreement with the speeds of the lenses used with the two systems. For the variable potential method, the patient dose ratio varied between 2.4 and 2.9.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Forty-seventh Annual Meeting: Roosevelt Hotel, New Orleans, La., April 8–10, 1965.

EDITORIAL

BARIUM OR AIR FOR EXAMINATION OF COLON

ROENTGENOLOGISTS have been entertained recently by a lively controversy about technical methods of roentgenologic examination of the colon. On the one hand are those who say in effect, "You err with air." On the other are some who reply in effect, "You blunder with barium." This difference of opinion has a double usefulness. It is making the diagnostic roentgenologist look at his technique with a more critical eye and consider his diagnoses with greater attention. Its second usefulness is to arouse the curiosity and therefore the greater interest of referring clinicians who gain more understanding of the examination.

In reappraising our efforts in the light of the present controversy, we are likely to decide that each of the extreme viewpoints has its advantages. We may further conclude that the truth, as it often does, lies somewhere in between. For most of us, the truth in this context means the most desirable method of examination in the daily work of the average roentgenologist.

Our present methods of radiologic examination of the colon have evolved with the development of our specialty. Stevenson[1] reviewed them 10 years ago. Opaque barium enema examination has been in use for more than 50 years, and the double contrast method was introduced more than 40 years ago. Many thoughtful, ingenious examiners have tried infinite variations in technique and improved the materials in use. Yet the basic ingredients of examination have remained. It would be surprising indeed if, without some completely new, major development, one of the long accepted standards of examination were suddenly discarded as useless.

Many lesions of the colon can be so strikingly demonstrated by the barium method that most radiologists have favored it as the basic standard procedure in the past. This choice seems likely to continue. Important factors for a successful examination include proper preparation of the patient, finesse in performance, good spot roentgenograms, high kilovoltage technique, immediate appraisal of roentgenograms, and varying the procedure according to the individual problem.

Too many roentgenologists have seen extraordinary demonstration of polyps and other lesions in air contrast examinations to advocate discarding this method. Claims of diagnostic errors may be correct, but errors occur with all types of examination. It is my belief that this is a useful supplementary examination. If the only lesion suspected is polyps, I would use it as the primary method.

It is somewhat ironic that while radiologic examiners have focused on the problem of identifying smaller and smaller polyps, some pathologists have been denying their importance. This pathologic controversy will doubtless continue. The radiologists' attention should not be diverted from attempting as refined a diagnosis as possible.

That physicians other than radiologists have become interested in our methods is good. Many physicians seem to have little concept of the factors that go into adequate roentgenologic examination of the colon. The considerable difference in quality is scarcely appreciated. Whereas some examinations and interpretations are relatively easy, in others performance is difficult and evaluation puzzling. It is our aim to make each examination as diagnostically useful as possible.

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[1] Stevenson, C. A. The development of the colon examination. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1954, *71*, 385–397.

In my opinion, several types of examination are helpful. The barium enema is considered the most valuable basic examination. Barium and air contrast examination seems a useful supplementary procedure, especially if the diagnosis of polypoid disease requires verification. In occasional patients, certain variations in technique may prove desirable, such as use of a balloon inflated in the rectum, fluoroscopy during or after evacuation, examination with the patient prone, or repeating the barium enema immediately after expulsion following the first enema.

The controversy about barium or air teaches that there are several methods of examination, each of which may have a particular usefulness. Why should we deny ourselves use of any method which may be helpful in some patients? The clinical roentgenologist should have a flexible attitude and a variable potential of examining techniques. He may have a standard method he prefers. If he encounters a diagnostic problem, however, he should be able to approach it with something more in his diagnostic quiver than a single methodical arrow.

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NEWS ITEMS

POSTGRADUATE COURSE IN PEDIATRIC RADIOLOGY

A Postgraduate Course in Pediatric Radiology with emphasis on selected special topics will be held at the Convention Center of the John Marshall Hotel, Richmond, Virginia, April 21–24, 1965. The course is being sponsored by the Department of Radiology of the Medical College of Virginia.

The guest faculty will include Dr. James B. Arey, St. Christopher's Hospital for Children, Philadelphia, Pa.; Dr. John A. Kirkpatrick, St. Christopher's Hospital for Children, Philadelphia, Pa.; Dr. Ralph V. Platou, Department of Pediatrics, Tulane Medical School, New Orleans, La.; Dr. Edward B. Singleton, Director of Radiology, St. Luke's and Texas Children's Hospital, Houston, Tex.; Dr. Eugene Van Epps, Department of Radiology, University Hospitals, Iowa City, Ia.; and Dr. M. H. Wittenborg, Children's Hospital Medical Center, Boston, Mass.

Further information may be obtained from Dr. M. Pinson Neal, Jr., Chairman, Postgraduate Course, Department of Radiology, Medical College of Virginia, Richmond, Va.

INTERNATIONAL ATOMIC ENERGY AGENCY

A Symposium on Radioisotope Sample Measurement Techniques in Medicine and Biology will be held in Vienna, Austria, on May 24–28, 1965, under the auspices of the International Atomic Energy Agency (IAEA).

Recent advances in methods of sample assay for alpha-, beta-, and gamma-ray emitters and technical progress in instrumentation for such measurements will be discussed.

Requests to participate and/or to present papers must be submitted through the appropriate national authorities responsible for atomic energy matters from whom detailed information and application forms can be obtained.

For further information contact Dr. Gerald J. Hine, Ph.D., Division of Isotopes, Section of Medicine, International Atomic Energy Agency, Kaerntnerring, Vienna I, Austria.

BOOK REVIEWS

Books sent for review are ackonwledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Year Book of Radiology: 1963–1964 Series. Radiologic Diagnosis. Edited by John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan; and Walter M. Whitehouse, M.D., Professor, Department of Radiology, University of Michigan. Radiation Therapy. Edited by Harold W. Jacox, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia University; Chief, Radiation Therapy Division, Radiologic Service, Presbyterian Hospital, New York, N. Y.; and Morton M. Kligerman, M.D., Professor of Radiology and Chairman of the Department of Radiology, Yale University School of Medicine; Radiologist-in-chief, Grace-New Haven Community Hospital. Cloth. Pp. 432, with 332 illustrations. Price, $11.75. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago 1, Ill., 1964.

It is extremely stimulating to note how each Year Book of Radiology seems to surpass its predecessor in valuable material and general make-up. The student, the resident, the practicing radiologist, and any physician interested in scientific medicine will find the 1963–1964 Edition delightful to read and useful in his daily endeavors. The most outstanding articles of world medical literature in this field are abstracted, condensed and presented with accompanying radiographic reproductions, illustrative drawings, and candid editorial comment.

Drs. Holt and Whitehouse have emphasized the increasing usefulness of contrast medium studies which seem to involve almost every organ system in the body. Rapid advances in television technology, cineradiography, and television tape recording are reviewed. Radiologic changes in disease of genetic origin are stressed, with articles on trisomy 17-18 syndrome, hypophosphatasia, cleidocranial and metaphysial dysostoses, and other rare conditions. The reader is referred to original articles whenever the reviewers deem it necessary.

One is impressed with the sections dealing with mammography, colonic disease, gallbladder physiology, and urogenital radiology, while cerebral and coronary arteriography continue to be objects of many clinical investigations.

In the therapeutic section Drs. Jacox and Kligerman have reviewed the changing concepts in the treatment of various diseases including pituitary adenomas, breast cancer, and gynecologic malignancies. Six articles on electron beam therapy are reviewed. The increasingly complex fields of radiobiology, isotope therapy, and radiologic physics are represented, and a section is devoted to hazards and injuries.

The editors have accomplished a tremendous task in selecting the most useful and important articles which are scientifically sound or denote progress, distinguishing them from fads or results of superficial thinking. We should realize our great privilege in enjoying at our leisure the fruits of their labor.

Arch H. Hall, M.D.

An Atlas of Vascular Rings and Related Malformations of the Aortic Arch System. By James R. Stewart, M.D., M.S. in Radiology; Consultant, Section of Roentgenology, Mayo Clinic, Rochester, Minnesota; Owings W. Kincaid, M.D., M.S. in Radiology; Consultant, Section of Roentgenology, Mayo Clinic; Assistant Professor of Radiology, Mayo Foundation Graduate School, University of Minnesota, Rochester, Minnesota; and Jesse E. Edwards, M.D.; Presently, Director of Laboratories, Charles T. Miller Hospital, St. Paul, Minnesota; Clinical Professor of Pathology, School of Medicine, University of Minnesota, Minneapolis, Minnesota. Cloth. Pp. 172, with many illustrations. Price, $10.75. Charles C Thomas, Publisher, 301–327 East Lawrence Ave., Springfield, Ill., 1963.

A monograph, according to the Merriam-Webster Collegiate Dictionary, is "A written account of a single thing or class; a special treatise on a particular subject." The ideal monograph, one might believe, should be clear, precise, complete, brief, fully illustrative of the subject under consideration and well written.

Using these criteria, "An Atlas of Vascular

Rings and Related Malformations of the Aortic Arch System" is as nearly ideal a monograph as can reasonably be expected. Following a concise description of the embryology of the aortic arch system, each of the forms of vascular rings and associated malformations of the aortic arch system are demonstrated.

Precise anatomic definition of these malformations is of particular importance to the radiologist and the surgeon. Because of the multiplicity of anatomic variations resulting from anomalous development of the aortic arch system, many of these are poorly understood. Careful study of this book should result in a complete understanding of the subject matter and permit precise diagnosis of these conditions.

The photographs of the gross pathologic specimens are exceptional. Each syndrome is fully illustrated. The specimens are clearly marked and easy to understand. The conventional roentgenologic material is also good and illustrates the various types of vascular rings. There is unfortunately a relative paucity of aortographic and angiocardiographic illustrative material, but this weakness is greatly overshadowed by the wealth of other information presented, in both visual and textually documentative form.

This monograph is highly recommended to all radiologists. It is well written, concise, and lucidly illuminates the subject.

Richard G. Lester, M.D.

Encyclopedia of X-Rays and Gamma Rays. Edited by George L. Clark, Research Professor of Analytical Chemistry, Emeritus, University of Illinois, Urbana, Ill. Cloth. Pp. 1149, with many illustrations and tables. Price, $35.00. Reinhold Publishing Corporation, 430 Park Avenue, New York 22, N. Y., 1963.

My first serious introduction to the world of x-rays came through Dr. Clark's book, "Applied X-rays." Now, more than 25 years later, Editor Clark leads a veritable army of 300 authors to present a panorama of equipments, techniques, methods and applications of x- and gamma-radiations through more than 350 concise articles. His high standard of editorship is evident in the constant level of sophistication maintained throughout the volume. One of the most interesting articles, of which he is the author, is a chronology of events from the year 1600 to the present. There is an excellent group of articles on dosimetry. The quality of the illustrations is very high.

In spite of the breadth of coverage of the many uses of x-ray equipment in chemistry, physics, engineering, etc., it will surprise the many radiologist readers to find that reference to their profession is almost completely absent. In fact, the word "radiology" is not found in the index. There are only four very brief articles on "medical radiography" and one on "radiation therapy"; each of these concern exotic techniques—none are fundamental reviews as are given for the other x-ray specialties. Fluoroscopy is discussed only from the industrial viewpoint. Dental radiographic technique is quite incompletely covered. The four page article on "Shielding" concerns itself only with fallout protection. The widespread use of cobalt 60 in teletherapy sources and industrial radiography merits more than a 17-word reference in the article on "Generation of X-rays."

Included are a number of excellent review articles exceeding 20 pages in length on such subjects as "Information Theory," "Image Intensification," and "High Temperature X-ray Diffraction."

The reviewer also was impressed with the paucity of printing errors. A system of cross-references is used to advantage throughout the text.

This volume should be of interest to physical scientists and technicians in industrial analytical laboratories.

Charles S. Simons, Ph.D.

BOOKS RECEIVED

Anatomico-Roentgenographic Studies of the Spine. By Lee A. Hadley, M.D., Senior Attending Roentgenologist, Syracuse Memorial Hospital, Syracuse, N. Y.; Lecturer in Radiology, Upstate Medical Center, New York State University, Syracuse, N. Y.; Former Clinical Associate Professor of Public Health, Syracuse University College of Medicine, Syracuse, N. Y. Cloth. Pp. 545, with many illustrations. Price, $26.00. Charles C Thomas, Publisher, 301–327 E. Lawrence Ave., Springfield, Ill., 1964.

Directory of Whole-Body Radioactivity Monitors: (Low Activity Levels). Cloth. Pp. 704, with illustrations and many tables. Price, $14.00. International Atomic Energy Agency, Vienna. In the U.S.A., International Publications, Inc., New York 16, N. Y., 1964.

Zerebrale Angiographie beim Frischen Schädel-Hirn-Trauma. Von Priv.-Doz. Dr. med.

Peter Huber, Neurochirurgische Abteilung der Chirurgischen Universitäts-Klinik Bern; mit einem Geleitwort von Prof. Dr. med. A. Zuppinger, Direktor des Röntgeninstitutes und der Radiotherapeutischen Abteilung des Inselspitals und der Universität Bern. Cloth. Pp. 109, with many illustrations. Price, DM 48. Georg Thieme Verlag, Postfach 732, Herdweg 63, 7000 Stuttgart 1, Germany, 1964.

Report of the Commission on Drug Safety. Cloth. Pp. 228. Price, $5.00. Federation of American Societies for Experimental Biology, 9650 Wisconsin Avenue, N.W., Washington, D. C. 20014, 1964.

National Cancer Institute Monograph 14. International Symposium Control of Cell Division and the Induction of Cancer. Edited by C. C. Congdon, M.D., and Pablo Mori-Chavez, M.D.; with technical assistance of the editorial staff, Biology Division, Oak Ridge Laboratory, Oak Ridge, Tennessee. Cloth. Pp. 403, with illustrations and many tables. Price, $4.50. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, 1964.

Fundamentals of Roentgenology. By Lucy Frank Squire, M.D., Formerly Clinical Assistant Professor of Radiology, University of Rochester School of Medicine. Cloth. Pp. 363, with many illustrations. Price, $12.50. Harvard University Press, Cambridge, Mass., 1964.

Syndromes of the Head and Neck. By Robert J. Gorlin, D.D.S., M.S., Professor and Chairman, Oral Pathology, University of Minnesota; Jens J. Pindborg, D.D.S., Dr. Odont., Professor and Chairman, Oral Pathology, Royal Dental College, Copenhagen, Denmark; with a foreword by Victor A. McKusick, Professor of Medicine, Johns Hopkins Medical School. Cloth. Pp. 580, with many illustrations. Price, $18.50. McGraw-Hill Book Company, New York 36, N. Y., 1964.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Radisson Hotel, Minneapolis, Minn., Sept. 29–Oct. 2, 1964.

American Radium Society
Secretary, Dr. John L. Pool, 444 East 68th Street, New York, N. Y. 10021. Annual meeting: Roosevelt Hotel, New Orleans, La., April 8–10, 1965.

Radiological Society of North America
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 29–Dec. 4, 1964.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Bellvue Stratford Hotel, Philadelphia, Pa., Feb. 10–13, 1965.

Section on Radiology, American Medical Association
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: New York, N. Y., June 20–24, 1965.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn. The Fall 1964 examination will be held at the Americana Hotel, Bal Harbour, Florida, December 7–11, inclusive. Deadline for filing applications was June 30, 1964.

The Spring 1965 examination will be held at the San Francisco Hilton Hotel, San Francisco, Calif., June 13–18, inclusive. This will be the last time that the Special Examination in Nuclear Medicine will be offered to physicians who are already certified in Radiology or Therapeutic Radiology. The deadline for filing applications for the above two examinations is December 31, 1964.

The Fall 1965 examination will be held at the Statler Hilton Hotel, Dallas, Texas, December 5–10, inclusive. The deadline for filing applications for this examination is June 30, 1965.

American Association of Physicists in Medicine
Secretary-Treasurer, Charles S. Simons, University of Michigan Hospital, Ann Arbor, Mich. Annual meeting to be announced.

American Club of Therapeutic Radiologists
Secretary, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo.

Eleventh International Congress of Radiology
Secretary-General, Professor Dr. Med. Arduino Ratti, via Moscova, 44-1, Milan, Italy. Address inquiries to Professor Dr. Med. Luigi Turano, President-Elect, Istituto di Radiologia, Università di Roma, Rome, Italy. Meeting: September 22–28, 1965.

Eighth Inter-American Congress of Radiology
Counselor for the United States, Dr. Philip J. Hodes, Jefferson Medical College Hospital, 11th and Walnut Streets, Philadelphia 7, Pennsylvania.
Secretary-General, Dr. Rubén Merenfeld, P.O. Box 9362, Caracas. Meeting: Caracas, Venezuela, Oct. 2–9, 1964.

Alabama Radiological Society
Secretary, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

American Nuclear Society
Treasurer, Raymond Maxson, 86 E. Randolph St., Chicago, Ill. Annual meeting: San Francisco, Calif., Nov. 30–Dec. 3, 1964.

Arizona Radiological Society
Secretary-Treasurer, Dr. George Gentner, 3435 W. Durango, Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Alexander R. Margulis, Department of Radiology, University of California, San Francisco, Calif. Annual meeting: Seattle, Washington, May 14 and 15, 1965.

Atlanta Radiological Society
Secretary, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Dr. Roy R. Deffebach, Major, MC, Radiology Service, 5th General Hospital, APO 154, New York, N. Y. Meets quarterly.

Blockley Radiological Society
Secretary, Dr. Bernard J. Ostrum, 2412 North 52nd St., Philadelphia, Pa.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Daniel J. Hanson, University of Kentucky, Medical Center, Lexington, Kentucky. Meets quarterly.

Brooklyn Radiological Society
Secretary, Dr. Jay P. Sackler, Brooklyn, N. Y. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Berkeley Zinn, 85 Wehrle Dr., Buffalo 25, N. Y. Meets second Monday evening each month, October to May inclusive.

California Radiological Society
Secretary, Dr. L. Henry Garland, Suite 1739, 450 Sutter St., San Francisco, Calif. Meets annually during meeting of California Medical Association.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P.O. Box 303, Rutherford College, N. C. Meets every Tuesday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C. at 12:00 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Edward W. Carsky, Crouse-Irving Hospital, 820 S. Crouse Ave., Syracuse, N. Y. Meets first Monday each month, October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Harold W. Long, 81 S. Fifth St., Columbus 15, Ohio. Meets third Thursday in October, and second Thursday in November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary-Treasurer, Dr. Robert D. Moseley, Jr., Dept. of Radiology, Univ. of Chicago, 950 E. 59th St., Chicago 37, Ill. Meets second Thursday of each month, October to April except December at the Pick-Congress Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary-Treasurer, Dr. James Christie, 10515 Carnegie

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* **Secretaries of societies are requested to send timely information promptly to the Editor.**

Avenue, Cleveland, Ohio. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society

Secretary, Dr. David J. Stephenson, 8300 West 38th Ave., Wheat Ridge, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society

Secretary, Dr. William W. Walthall, Jr., 137 Maple St., Hartford 6, Conn. Meets in April and October.

Dallas-Fort Worth Radiological Society

Secretary, Dr. Paul W. Mathews, Jr., 1217 West Cannon St., Fort Worth, Tex. Meets monthly, third Monday, at Southwest International Airport at 6:00 P.M.

Detroit Roentgen Ray and Radium Society

Secretary, Dr. Arch H. Hall, Harper Hospital, Detroit 1, Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society

Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland, Calif.

East Tennessee Radiological Society

Secretary, Dr. J. Marsh Frere, Jr., 205 Medical Arts Bldg., Knoxville, Tenn. Meets in January and September.

Eastern Conference of Radiologists

Secretary, Arrangements Committee, Dr. Theodore A. Tristan, 3400 Spruce St., Philadelphia 4, Pa. Annual meeting to be announced.

Eastern Radiological Society

Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.

Florida Radiological Society

Secretary, Dr. David Kirsh, 712 Du Pont Bldg., Miami, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society

Secretary-Treasurer, Dr. Garth R. Drewry, Tampa General Hospital, Tampa 6, Fla. Meets in January, April, July and October.

Georgia Radiological Society

Secretary, Dr. David Robinson, P.O. Box 394, Savannah, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society

Secretary-Treasurer, Dr. Oliver P. Winslow, Jr., Baptist Hospital of Miami, Inc., 8900 S. W. 88th St., Miami 56, Fla. Meets monthly, third Wednesday at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Society of Radiologists

Secretary, Dr. C. M. Witt, #16 Hampton Village Plaza, St. Louis 9, Mo.

Houston Radiological Society

Secretary, Dr. D. A. Van Velzer, Texas Medical Center Library, Jesse H. Jones Library Bldg., Houston 25, Tex. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 P.M., Houston, Tex.

Idaho State Radiological Society

Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

Illinois Radiological Society

Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

Indiana Roentgen Society, Inc.

Secretary, Dr. Richard A. Silver, 1815 N. Capitol Avenue, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society

Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society

Secretary-Treasurer, Dr. R. F. Conard, Newman Memorial Hosp., Emporia, Kan. Meets in spring with State Medical Society and in winter on call.

Kentucky Radiological Society

Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Building, Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville, Ky.

Kings County Radiological Society

Secretary, Dr. Stanley Dannenberg, 1917 Bedford Ave., Brooklyn 25, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society

Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Los Angeles Radiological Society

Secretary, Dr. Austin R. Wilson, 540 N. Central Ave., Glendale, Calif. 91203. Meets second Wednesday of month in November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Maine Radiological Society

Secretary, Dr. J. T. Chen, 7 Cherry Hill Terrace, Waterville, Me. Meets in June, September, December and April.

Maryland Radiological Society

Secretary, Dr. Henry Startzman, Medical Arts Building, Baltimore, Maryland.

Memphis Roentgen Society

Secretary-Treasurer, Dr. Irving K. Ettman, 684 W. Brookhaven Circle, Memphis, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society

Secretary, Dr. William D. Roberts, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society

Secretary-Treasurer, Dr. Alexander W. Friedman, Mid-Hudson Medical Group, Fishkill, N. Y. Meets 7:00 P.M., first Wednesday of each month September to May.

Milwaukee Roentgen Ray Society

Secretary-Treasurer, Dr. Harold F. Ibach, 2400 W. Villard Avenue, Milwaukee 9, Wis. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society

Secretary-Treasurer, Dr. Frank J. Anderson, 810 E. 27th St., Minneapolis 7, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society

Secretary-Treasurer, Dr. Dan T. Keel, Jr., 504 Chippewa St., Brookhaven, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society

Secretary-Treasurer, Dr. M. Shoss, Cape Girardeau, Mo.

Montana Radiological Society

Secretary, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.

Nassau Radiological Society

Secretary, Dr. Barry L. Lewis, 165 N. Village Ave., Rockville Centre, New York, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska State Radiological Society

Secretary, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society

Secretary, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

New England Roentgen Ray Society

Secretary, Dr. Jack R. Dreyfuss, Zero Emerson Place, Boston, Mass. 02114. Meets third Friday of each month, October through April, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New Mexico Association of Radiologists
Secretary-Treasurer, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

New York Roentgen Society
Secretary, Dr. Harry Z. Mellins. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. John Jestadt, Depuy-Sorkness Clinic, Jamestown, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. John M. Johannessen, Mercy Hospital, Sacramento, Calif. Meets fourth Monday of Sept., Nov., Jan., March and May at the Sutter Club in Sacramento.

Northwestern Ohio Radiological Society
Secretary. Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society
Secretary, Dr. Chapin Hawley, Dept. of Radiology, University of Cincinnati College of Medicine, Cincinnati, Ohio. Annual meeting: Terrace Hilton Hotel, Cincinnati, Ohio, May, 1965.

Oklahoma State Radiological Society
Secretary, Dr. Dave B. Lhevine, B10 Doctors Bldg., 2021 South Lewis, Tulsa, Okla. Meets in January, May and October.

Orange County Radiological Society
Secretary-Treasurer, Dr. Robert M. Sodaro, 600 E. Washington Ave., Santa Ana, Calif. Meets fourth Tuesday of month except June, July, August and December in Orange County Medical Association Building.

Oregon Radiological Society
Secretary-Treasurer, Dr. James A. Schneider, St. Vincent Hospital, Portland 10, Ore. Meets on second Wednesday of month at 7:00 P.M. at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. Willis Taylor, Seattle, Washington. Annual meeting to be announced.

Pennsylvania Radiological Society
Secretary-Treasurer, Dr. Frederick R. Gilmore, Clearfield Hospital, Clearfield, Pa. Annual meeting: Pittsburgh Hilton Hotel, May 21–22, 1965.

Philadelphia Roentgen Ray Society
Secretary, Dr. Antolin Raventos, Hospital of the University of Pennsylvania, Philadelphia 4, Pa. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Robert N. Berk, 3305 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Society of Connecticut
Secretary-Treasurer, Dr. Orlando F. Gabriele, 1450 Chapel St., New Haven 11, Conn. Meetings are held bimonthly.

Radiological Society of Greater Cincinnati
Secretary, Dr. John F. Popkin, Jr., 831 Carew Tower, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Hawaii
Secretary, Dr. Edgar S. Childs, Kuakini Hospital, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Andrew F. Giesen, Ochsner Clinic, New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. E. Arthur Kratzman, 912 Prospect Ave., Plainfield, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary-Treasurer, Dr. John L. Gwinn, Children's Hospital of Los Angeles, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Meets three times a year, usually October, February, and May.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, August, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Gerald R. Holzwasser, Rochester General Hospital, Northside Division, 1425 Portland Ave., Rochester 21, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting to be announced.

San Antonio-Military Radiological Society
Secretary, Dr. Hugho F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Charles P. Hyslop, 7901 Frost St., San Diego 22, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. Malcolm Jones, University of California Hospital, San Francisco 22, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Martin A. Thomas, 1150 Connecticut Ave., Washington 36, D. C. Meets at Medical

Society Library, third Wednesday of January, March, May and October at 8:00 p.m.

Section on Radiology, Southern Medical Association

Secretary,* Dr. John D. Reeves, Department of Radiology, Univ. of Florida College of Medicine, Gainesville, Fla. Annual meeting: Memphis, Tennessee, Nov. 16–19, 1964.

Section on Radiology, Texas Medical Association

Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club

Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 p.m., September to May inclusive.

Society for Pediatric Radiology

Secretary, Dr. John L. Gwinn, Children's Hospital, Los Angeles 27, Calif. Annual meeting: Radisson Hotel, Minneapolis, Minn., Sept. 28, 1964.

Society of Nuclear Medicine

Secretary, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting: Bal Harbour, Fla., June 16–19, 1965.

South Bay Radiological Society

Secretary, Northern Section: Dr. John H. Callaghan, 2900 Whipple Ave., Redwood City, Calif.; Southern Section: Dr. Paul L. Clemetson, 877 W. Fremont Ave., Suite C-3, Sunnyvale, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society

Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

Southern Radiological Conference

Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting: Grand Hotel, Point Clear, Ala., Jan. 29–31, 1965.

Southwestern Radiological Society

Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 p.m. in the Paso del Norte Hotel.

Tennessee Radiological Society

Secretary, Dr. B. M. Brady, St. Joseph Hospital, Memphis, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society

Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Tex. Annual meeting: Houston, Texas, Jan. 29–30, 1965.

Tri-State Radiological Society

Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 p.m., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting

Meets each Monday evening from September to June, at 7:00 p.m. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society

Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society

Secretary, Dr. Carlisle C. Smith, Salt Lake General Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society

Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

Virginia Radiological Society

Secretary, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va.

Washington State Radiological Society

Secretary, Dr. Owen Marten, 920 Terry Avenue, Seattle, Wash. Meets quarterly.

West Virginia Radiological Society

Secretary, Dr. Karl J. Myers, The Myers Clinic-Broaddus Hospital, Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society

Secretary, Dr. Joel J. Schwartzman, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society

Secretary, Dr. Charles Benkendorf, 408 St. Francis St., Green Bay, Wis. Annual meeting each spring in various places.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.

Secretary-General, Dr. Raul Arguello, 5a. Ave., Norte No. 48, San Salvador, El Salvador. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador

Secretary, Dr. Rafael Vaga Gómez.

Sociedad de Radiología de Guatemala

Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana

Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia

Secretary, Dr. James Fernández Carballo, Apartado viii, San José, Costa Rica.

Sociedad Mexicana de Radiología, A.C.

Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández.
Meets first Monday of each month.

Asociación Puertorriqueña de Radiología

Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña

Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico

Secretary, Dr. Jorge Carreras Girard, Suite 504, Professional Bldg., Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 p.m. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec

Secretary, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.

British Institute of Radiology

Honorary Secretary, Dr. R. D. Hoare, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics.

Honorary Secretary-Treasurer, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario, London, Ont., Canada. Annual meeting: Toronto, Ontario, Feb., 1965.

Edmonton and District Radiological Society

Secretary, Dr. J. T. Mason, 540 Tegler Bldg., Edmonton, Alberta, Canada. Meets second Tuesday of each month, October to May.

Faculty of Radiologists

Honorary Secretary, Dr. C. J. Hodson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

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* Deceased.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. D. J. Sieniewicz, *Associate Honorary Secretary-Treasurer*, Dr. Maurice Dufresne 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Maurice Dufresne, 1560 Sherbrooke (East), Montreal, Que., Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. Wallace M. Roy, St. Joseph's Hospitals, 30 The Queensway, Toronto 3, Ont., Canada. Meets second Monday of each month September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Tede Eston de Eston, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiologia
Secretary, Dr. Armando B. de Onaindia, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Armando Uribe, Hospital Miltar Central, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Luis Blum, P.O. Box 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Vicente Ubillus, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. Annual meeting to be announced.

Société Belge de Radiologie
General Secretary, Prof. Simon Masy, Louvain, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 8^e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut., Lagenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. H. F. O. Stricker, Schalkklaar, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

Indonesian Radiological Society
Secretary, Professor Sjahriar Rasad Taman Tjut Mutiah 1, Djakarta, Indonesia.

ABSTRACTS OF RADIOLOGICAL LITERATURE

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Head

Nahoum, H. I., Fiasconaro, J., and DiSalvo, N. A.: The verticosubmental roentgenogram in cephalometrics........................ 1212

Neck and Chest

Jesserer, H., *et al.:* The adaptability of selective angiography in the localization of parathyroid tumors........................ 1212
Adams, H. D.: Amyenteric achalasia of the esophagus........................ 1212
Robinson, R. G.: Intrathoracic meningocele and neurofibromatosis........................ 1212
Graham, A. J., *et al.:* Further experiments on long term survivors after circulatory bypass of the right side of the heart........................ 1213

Abdomen

Moldenhauer, W., and Reinck, H.: Parietography, an additional study of the stomach... 1213
Stempien, S. J., *et al.:* Hypertrophic hypersecretory gastropathy: analysis of 15 cases and a review of the pertinent literature.... 1214
Elebute, E. A., and Adesola, A. O.: Intussusception in Western Nigeria........................ 1214
Righi Riva, G. C., *et al.:* Differential diagnosis of "large irregular liver" by "semiologic triad": radiologic examination with pneumoperitoneum, etc. 1215
Mosca, L. G., and Croppi, C. J.: Accelerated cholecystography........................ 1215
Valenzuela, G. A.: Ascariasis of the biliary pathways and digestive tract........................ 1215

Genitourinary System

Diethelm, L., *et al.:* The choice of contrast material and the possibilities of double contrast visualization of the urinary bladder... 1216
Desgrez, H., *et al.:* Retrograde urethrography by flexible sound with balloon........................ 1216

Skeletal System

Bonte, J., *et al.:* Osteolysis after contusion. Three cases of osteolysis of the acromial end of the clavicle........................ 1216
Jones, W. C., III, and Miller, W. E.: Skeletal tuberculosis—1963........................ 1216
Rosenberg, N. J.: Osteochondral fractures of the lateral femoral condyle........................ 1217
Greville, N. R.: Osteochondritis dissecans: treatment by bone grafting........................ 1217
Tapia, J., Stearns, G., and Ponseti, I. V.: Vitamin-D resistant rickets: a long-term clinical study of eleven patients........................ 1217
Accomazzi, F., and Gaudino, F.: Considerations on the radiographic aspect of Cooley's disease in the adult........................ 1218
Blumenfeld, J. I.: Diabetic arthropathy: contribution to its study........................ 1218
Pitts, N. C.: Myositis ossificans as a complication of tetanus........................ 1218

Blood and Lymph System

Leger, L., and Mouktar, M.: Portal angiomegalies........................ 1218
Baird, R. J., and Doran, M. L.: The false aneurysm........................ 1219
Winckelmann, G., *et al.:* Thrombolytic treatment of acute arterial occlusion with streptokinase........................ 1219
Calnan, J. S., *et al.:* Venous obstruction in the aetiology of lymphoedema praecox........ 1219
Tilak, S. P., and Howard, J. M.: The influence of the dual circulation on the viability of lymph nodes following interruption of their blood or lymphatic supply........... 1220

General

Capotorti, L., Ferrante, E., and Bottini, E.: De Toni-Debré-Fanconi syndrome without demonstrable cystinosis in a 2 year old child........................ 1220
Moran, J. M.: An unusual foreign body..... 1221
Burrow, G. N., Hamilton, H. B., and Hrubec, Z.: Study of adolescents exposed *in utero* to the atomic bomb, Nagasaki, Japan. 1221

RADIATION THERAPY

Breed, J. E.: Radium therapy in cancer of the eyelid........................ 1222
Lee, E. S., and Mackenzie, D. H.: Osteosarcoma: a study of the value of preoperative megavoltage radiotherapy........................ 1222

RADIOISOTOPES

Sargent, T., *et al.:* Use of a whole body counter in turnover studies with Ca 47......... 1222
Rosenthall, L.: The use of strontium-85 for the detection of bone lesions........................ 1223
DiGiulio, W., and Beierwaltes, W. H.: Parathyroid scanning with selenium 75 labelled methionine........................ 1223

CHEMOTHERAPY

Greenberg, L. H., and Tanaka, K. R.: Congenital anomalies probably induced by cyclophosphamide........................ 1224
Speed, D. E., Galton, D. A. G., and Swan, A.: Melphalan in the treatment of myelomatosis........................ 1224

ROENTGEN DIAGNOSIS

Head

Nahoum, Henry I., Fiasconaro, Joseph, and DiSalvo, Nicholas A. (New York City.) The verticosubmental roentgenogram in cephalometrics. *J. Am. Dent. A.*, Aug., 1964, *69*, 133–150.

To obtain a more informative three dimensional image in roentgenographic cephalometry, the use of a verticosubmental projection is suggested in conjunction with the conventional lateral and posteroanterior projections. A Margolis cephalostat is employed to obtain these views and the technique of this procedure is described in detail.

Using a dry skull, measurements were made and compared with those determined from the roentgenograms. A table also shows the comparison of measurements projected horizontally from the lateral to the posteroanterior and those projected vertically from the posteroanterior to the verticosubmental.

Nine cases are reported in detail to demonstrate the value of this view.

This article should be read in the original by orthodontists and those radiologists who use cephalometrics.—*Martha Mottram, M.D.*

Neck and Chest

Jesserer, H., Kaindl, F. Kohn, P., and Kotscher, E. Über die Eignung einer gezielten Arteriographie zur Lokalisation von Nebenschilddrüsenadenomen. (The adaptability of selective angiography in the localization of parathyroid tumors.) *Radiologia Austriaca*, 1963, *14*, 305–308. (Address: Prof. Dr. Hans Jesserer, I. Medizinische Universitätsklinik, Lazarettgasse 14, Wein IX, Austria.)

Since extirpation of all intraglandular as well as aberrant parathyroid tumors is essential to obtain a cure in primary hyperparathyroidism, the authors employed Seldinger's method of selective angiography in an attempt to supply the surgeon with the maximum amount of preoperative information concerning the extent and location of such tumors. After insertion of a catheter via the brachial artery up to a point 1 cm. distal to the origin of the thyro-cervical trunk, opacification of the area was effected. The contralateral side was injected several days later.

This procedure was tried in 7 patients without a single successful outcome because some of the tumors were not identified by this method.

In this small series of cases, the authors found it often easier to localize aberrant tumors than eutopic lesions.—*J. Zausner, M.D.*

Adams, Herbert D. Amyenteric achalasia of the esophagus. *Surg., Gynec. & Obst.*, Aug., 1964, *119*, 251–256. (From: Department of Surgery, Lahey Clinic, Boston, Mass.)

The use of the term "achalasia" to denote all causes of a malfunctioning esophagus has obscured underlying etiology and frustrated attempts to find a single operative procedure for its treatment.

In amyenteric achalasia plain roentgenograms are frequently misleading but barium studies demonstrate the huge, dilated, saccular, malfunctioning esophagus. A simple, practical method of diagnosis, relatively free of hazard, which can differentiate amyenteric achalasia from cardiospasm or organic stricture is the passage of a large esophagoscope without obstructive resistance and the absence of significant relief from symptoms in spite of repeated dilatations of the lower end of the esophagus.

Any surgical procedure must correct two factors. The congenital aganglionic segment at the lower end of the esophagus and the large, dilated, saccular portion of the esophagus, which becomes angulated and obstructed when filled, must be removed.

The author observed 26 patients with amyenteric achalasia, ranging in age from 13 to 69 years, on whom an esophagogastric anastomosis immediately below the arch of the aorta, vagotomy, and pyloroplasty were performed. He presents appropriate diagrams showing the extent of the esophageal resection, including the lower sacculation and aganglionic segments.

Twenty-two of the patients having had the procedure are clinically well. There is no follow-up in 1 case, and 3 deaths have occurred due to pneumonia, ileocolitis, and cerebral thrombosis, respectively.

Meticulous surgery with preservation of function as a primary objective gives excellent results with low morbidity and no complications.—*Richard R. Oslund, M.D.*

Robinson, Richard G. Intrathoracic meningocele and neurofibromatosis. *Brit. J. Surg.*, June, 1964, *51*, 432–437. (From: Department of Neurosurgery, Dunedin Hospital, and Department of Surgery, University of Otago Medical School, Dunedin, New Zealand.)

Three cases of intrathoracic meningocele associated with neurofibromatosis are presented to bring the total cases reported with intrathoracic meningoceles to 47. Seventy per cent of these had had an associated neurofibromatosis.

Found equally in both sexes between the ages of 30 and 50 years, discovery is most frequently by routine chest roentgenograms where a round shadow projects laterally and posteriorly in the posterior mediastinum. The lesions are usually on the right side and occur most commonly above the sixth thoracic vertebra. Rib symmetry may be distorted and the intervertebral foramen is enlarged due to smooth erosion of the adjacent pedicles. Scalloping of the posterior margins of the vertebral bodies may occur, both above and below the lesion.

Differentiation from other posterior mediastinal tumors of the dumbbell variety in the absence of an associated neurofibromatosis depends upon the demonstration of dysplastic changes in the vertebral bodies, wedging of the vertebral bodies with smooth upper and lower surfaces, and the sparing of the intervertebral disks. The entire spine may be involved in some of the deformities and a severe scoliosis or kyphoscoliosis may be apparent. In the presence of neurofibromatosis without neurologic signs, a rounded shadow in the posterior mediastinum most probably represents a meningocele rather than a neurofibroma.

Laminagraphy helps evaluate the spinal changes.

Myelography with contrast filling of the meningocele is diagnostic and should be used in doubtful cases, where surgery is planned, or in the presence of neurologic signs. The author notes that most cases are static and do not require surgery.

It is emphasized that the bone abnormalities typical of intrathoracic meningocele may also occur in neurofibromatosis without meningocele. Lumbosacral meningoceles and myelomeningoceles in patients with neurofibromatosis are probably coincidental. Orbital meningoceles in patients with neurofibromatosis are similar to intrathoracic meningoceles.—*Richard R. Oslund, M.D.*

GRAHAM, ANDREW J., RICKETTS, HOWARD J., FENN, JOHN E., LARSEN, PARRY B., CEV, MUZAFFER, and GLENN, WILLIAM W. L. Further experiments on long term survivors after circulatory bypass of the right side of the heart. *Surg., Gynec. & Obst.*, Aug., 1964, *119*, 302–310. (From: Department of Surgery, Yale University School of Medicine, New Haven, Conn.)

This is a follow-up study of 36 long term surviving animals with vena cava-pulmonary artery shunts. Twenty-six of these have had superior vena cava-right pulmonary artery shunt (SVC-RPA) performed and 10 inferior vena cava-right pulmonary artery shunt (IVC-RPA). The oldest surviving animal is now more than 9 years postoperative. No animal was included in the study which was less than 6 months postoperative.

The shunt between the superior vena cava and right pulmonary artery has continued to function well for 9 years, as shown by angiography. Shunt flow, measured at intervals for 6 years, showed no significant change. Venous pressure, elevated initially, fell rapidly during the first year postoperatively but was still not normal after 6 years.

In these experiments, collateral flow from the SVC to the IVC was commonly demonstrated by prominence of the pericardiophrenic vein. Normal cephalic flow in the vertebral and azygos veins was reversed by the superior vena cava shunt, and prominent lumbar veins connecting the two venae cavae were seen. Ligation of the inferior vena cava below the renal veins re-established flow in the normal direction, with some increase in the return to the superior vena cava. Any increase in the superior vena cava return was slight and transient; however, large vertebral and lumbar collateral channels developing around the inferior vena cava ligature returned blood to the inferior vena cava. Ligation of the latter, therefore, was not an effective method of increasing blood flow through the SVC-RPA anastomosis, and indeed, in these animals, appeared to promote development of uncontrollable chylothorax.

A patent azygos vein in conjunction with an SVC-RPA anastomosis diverted some of the superior vena cava blood through its tributaries to the inferior vena cava. Maximum flow through the shunt was, therefore, obtained by dividing the axygos vein at its junction with the superior vena cava.

The initial survival rate of animal subjects was poor because of pooling of blood in the splanchnic vessels. Postoperative flow studies in surviving animals indicated that only about one-half the blood normally returning through the inferior vena cava to the right atrium passed through the shunt. Chronic passive congestion was found in all animals as evidenced by persistent ascites and hepatomegaly.

Fairly detailed description of the methods of study and measurement are given by the authors.—*C. Peter Truog, M.D.*

ABDOMEN

MOLDENHAUER, W., and REINCK, H. Die Parietographie, eine Zusatzuntersuchung des Magens. (Parietography, an additional study of the stomach.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1964, *100*, 569–578. (Address: Dr. med. W. Moldenhauer, Facharzt für Röntgenologie, Oberarzt und Leiter der Röntgen-Abteilung der Mediz. Univ.-Klinik Rostock, Ernst-Heydemann-Strasse, Rostock, Germany.)

Finer details of the gastric wall, particularly small zones of minimal thickening, can be demonstrated by means of parietography. A pneumoperitoneum is achieved by instillation of 1,000–1,500 cc. of oxygen in a period of one-quarter hour. The stomach is distended by the use of an effervescent powder (rather than an intragastric balloon, which is often uncomfortable for the patient). Tomography with the simultaneous exposure of 7 films is performed in the anteroposterior direction but may be supplemented by the lateral or oblique projections. Simultaneous tomography affords the advantage of being able to view the stomach in the same peristaltic phase.

The indications for this procedure are: (1) when conventional methods fail; (2) for tomographic definition of a tumor's extent; (3) to rule out the presence of a tumor; and (4) when gastric bleeding interferes with a satisfactory examination. (Incidentally, the stomach should not be distended in the presence of an acute gastric ulcer with bleeding or perforating tendency.)

The contraindications to pneumoperitoneum are the usual ones; *i.e.:* (1) acute inflammatory conditions of the abdomen; (2) extensive intra-abdominal adhesions; (3) anomalies of the diaphragm; (4) decompensated heart disease; and (5) respiratory distress.

This method was employed in 44 patients to provide the surgeon with valuable information concerning the intra- and extra-gastric extent of the lesions. Metastatic nodes and lesions on the hepatic surface were frequently identified. Thus the inoperability of a gastric lesion could be more readily determined. The differential diagnosis between benign and malignant ulcers is materially aided by this study. The detailed study of tumors of the gastric fundus is considerably facilitated. Small lesions confined to the gastric mucosa are difficult to diagnose. A thorough anatomic knowledge of the various gastric attachments is essential, otherwise they may be misinterpreted as adhesions.—*J. Zausner, M.D.*

Stempien, S. J., Dagradi, A. E., Reingold, I. M., Heiskell, C. L., Goodman, J. R., Bloom, A., and Weaver, D. S. Hypertrophic hypersecretory gastropathy: analysis of 15 cases and a review of the pertinent literature. *Am. J. Digest. Dis.*, July, 1964, *9*, 471–493. (Address: Veterans Administration Hospital, 5901 East 7th Street, Long Beach, Calif.)

The authors report and analyze their experience with 15 patients with a gastric mucosal disease previously called hypertrophic glandular gastritis. They believe this to be an important and well defined clinical and pathologic entity, which may exist alone or combined with duodenal ulcer, peptic ulcer associated with specific or multiple endocrinopathies, and the Zollinger-Ellison syndrome. There are 4 clinical forms: (a) intractable epigastric pain, (b) anorexia, nausea and vomiting without evidence of obstruction, (c) upper gastrointestinal hemorrhage, and (d) unexplained diarrhea.

Roentgenographic appearances alone are the least reliable as gastric mucosal hypertrophy may be due to other causes. In this disease there is marked hypersecretion of gastric HCl, pepsin and mucoprotein but no significant protein loss occurs into the gastric juice. The mucosal hypertrophy involves all the glandular elements, parietal cells, chief cells, and mucous cells. Peroral biopsy techniques are unreliable and serve only to rule out diffuse malignant changes. If any doubt as to the diagnosis exists, full-thickness surgical biopsy must be obtained at laparotomy. In most cases, however, diagnosis can be made from roentgenologic, gastroscopic and secretory findings without surgical biopsy.

The symptoms of this disease can be ameliorated with adequate vagotomy-pyloroplasty. In 2 of the authors' cases radiation therapy gave gratifying results, suggesting that this method of treatment should be further explored.—*Arthur E. Childe, M.D.*

Elebute, E. A., and Adesola, A. O. Intussusception in Western Nigeria. *Brit. J. Surg.*, June, 1964, *51*, 440–444. (From: Department of Surgery, University College Hospital, Ibadan, Nigeria.)

The authors report 95 cases of intussusception occurring in 94 patients between April, 1958, and March, 1962. Intussusception in Western Nigeria is not a disease confined to infancy but occurs frequently in older children and in adults. Fifty-one of their cases were males and 43 females; 16.8 per cent were infants below 1 year and 42 per cent were adults; *i.e.*, 15 years or older. The youngest patient was 3 months old and the oldest 59 years.

There was a seasonal incidence with an increase in the number of cases in November, December and January. The cause of the intussusception was known in only 5 of the 95 cases: in 2 it was caused by Meckel's diverticulum in 1 by benign fibroadenomatous polyp, in 1 by carcinoma of the cecum, and in 1 by ascariasis. Ninety of the cases were of the primary or idiopathic type of intussusception.

The apex of the intussusception was found most commonly in the transverse colon or in the left colon. In 3 cases the apex protruded well out of the anus. One of these patients was a girl of 8 years who required ileocolectomy. The other 2 cases, a 1 year old male infant and a 53 year old male, were treated easily by operative reduction of the intussusception. All 3 cases survived.

Over one-half of the cases were of the acute variety (1–7 days) but about one-fifth were chronic (over 2 weeks). The 4 cardinal clinical features of the intussusception were: abdominal colic; vomiting; passage of blood and mucus per rectum; and a palpable mass. These features were not always present together in one patient; 43 of the 95 cases (45 per cent) presented all the 4 features. The reduction of the intussusception was possible in 82 per cent of the cases; in the majority of these the reduction was easy; anchorage of the cecum was done in only 4 instances and no recurrence occurred. In 10 cases appendectomy was performed after reduction had been achieved. This was usually thought indicated when the condition of the appendix suggested severe inflammation or gangrene.

Bowel resection was performed in 16 per cent of the cases. In 2 of these, resection of small bowel was carried out for irreducible enteric intussusceptions; in 12 cases ileocolectomy was done and 2 cases were treated by the exteriorization-resection procedure of

Mikulicz. The operation was performed under general anesthesia in all but 3 patients, who were very ill and had to have abdominal field block with local anesthesia.

Postoperative diarrhea was troublesome in a few cases; 7 cases had this complication.

There were 6 deaths out of 95 cases—a mortality rate of 6.3 per cent. Three of these cases had other severe associated conditions. One patient, who had severe pulmonary tuberculosis and a massive left pleural effusion, died from postoperative pulmonary embolism. Another patient, whose intussusception was caused by a carcinoma of the cecum, had carcinomatosis involving all pelvic organs and both lungs. A third patient had typhoid septicemia.

Of the 3 patients whose deaths may be attributed to intussusception, 1 was treated by ileocolectomy and developed peritonitis from disruption of the anastomosis. The other 2 were infants who were gravely ill before operation. They were treated by reduction of the intussusception but died soon after the operation.

The authors state that they have found roentgenologic examination of no special value in the diagnosis of intussusception except in a minority of doubtful cases. They feel that most of the cases can be diagnosed purely on clinical grounds. They also feel that the treatment of choice for cecocecal intussusception seen in Western Nigeria is operative reduction, which is possible in most cases. They believe that reducibility does not coincide with the length of the history. Many of the chronic cases reduce easily and a few of the acute cases require bowel resection.—*C. Peter Truog, M.D.*

Righi Riva, G. C., Di Marco, G., Dardari, M., and Santino, A. Diagnostica differenziale del "grosso fegato irregolare" mediante la "triade semeiologica": esame radiologico con pneumoperitoneo, laparoscopia e biopsia epatica. (Differential diagnosis of "large irregular liver" by "semiologic triad": radiologic examination with pneumoperitoneum, laparoscopy and hepatic biopsy.) *Radiol. med.*, May, 1964, *50*, 401–429. (Address: Dott. Giancarlo Righi Riva, Via Monte Sabotino 99, Modena, Italy.)

The authors review the laparoscopic and pneumoperitoneum findings observed in processes producing the so-called large irregular liver.

In the posthepatic hepatosclerosis, there is evidence of multiple bulgings from the liver surface, usually smooth and regular, of variable size and involving the entire viscus or just one lobe. Splenomegaly is always present. At laparoscopy it is possible to observe through the capsule a normal lobular pattern of the hepatic parenchyma.

In the perihepatic hepatosclerosis, the typical finding is the diffuse adhesive perihepatitis producing a considerable fixation of the liver. At laparoscopy one observes the typical sugar candy-like appearance of the liver surface due to the involvement of the capsule.

Echinococcus cyst of the liver and polycystic liver produce one or multiple masses protruding from the liver surface. Of interest is the difference in color of the tumefaction observed at laparoscopy which is gray in parasitic cysts, whereas in polycystic liver the color of the cyst depends upon the type of fluid contained, which might be bloody, mucous or biliary.

The diagnosis of benign tumor is difficult except for the hemangioma which at laparoscopy appears as a large smooth mass of typical red color. Primary and secondary malignant neoplasms have an appearance ranging from aspecific bulging of the liver surface to typical masses producing infiltration of the capsule and of the surrounding viscera.

Laparoscopy has the unquestionable value of permitting selective hepatic biopsies.

The number of cases examined by pneumoperitoneum and laparoscopy and the eventual complications are not specified by the authors in this article.—*Galdino E. Valvassori, M.D.*

Mosca, Lidio G., and Croppi, Carlos J. La colecistografia acelerada. (Accelerated cholecystography.) *Radiologiá*, Panamá, June, 1964, *14*, 87–93. (From: Hospital Córdoba, Córdoba, Argentina.)

Factors affecting the rate of opacification of the gallbladder include gastric emptying, solubility of contrast medium in the intestinal tract, rate of choleresis, and ability of the gallbladder to concentrate the opaque medium. Biloptin was found to more readily absorb than telepaque. Cold water and morphine (preferably given orally as paregoric) accelerate gastric emptying and morphine closes the sphincter of Oddi, thus aiding opacification of the biliary system. Nonsynthetic bile acids enhance choleresis.

Following administration of 6 capsules of biloptin (oragrafin in U.S.) and 4 tablets of dehydrocholic acid, with 4 more tablets of dehydrocholic acid 30 minutes later, it was found that a satisfactory cholecystogram could be secured in 2 hours in 70 per cent of the patients and in 3–4 hours in the other 30 per cent.—*Charles M. Nice, Jr., M.D., Ph.D.*

Valenzuela, German Abad. (Guayaquil, Ecuador.) Ascaridiasis de las vias biliares y del tubo digestivo. (Ascariasis of the biliary pathways and digestive tract.) *Radiologiá*, Panamá, June, 1964, *14*, 99–104.

A high percentage of patients in Ecuador have ascariasis involving the intestinal tract. The findings of filling defects in the barium study of the small bowel and the finding of a long, narrow "string" of barium in the intestinal tract of the ascaris itself are both well known signs. A pseudopolypoid appearance may be noted in the duodenal bulb and upper portion of the loop.

With the use of intravenous cholangiography the worms may be demonstrated in the common bile duct in patients who are having intense colic, which has not yielded to the usual antispasmotics and analgesics.—*Charles M. Nice, Jr., M.D., Ph.D.*

GENITOURINARY SYSTEM

DIETHELM, L., RAPP, W. J., VITA, G., and ZEITLER, E. Kontrastmittelwahl und Möglichkeiten bei Doppelkontrastdarstellung der Harnblase. (The choice of contrast material and the possibilities of double contrast visualization of the urinary bladder.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, June, 1964, *100*, 727–736. (Address: Prof. Dr. med. L. Diethelm, Dir. am Univ.-Inst. für Klinische Strahlenkunde, 65 Mainz, Langenbeckstrasse 1, Mainz, Germany.)

The authors avoid the use of air or oxygen for double contrast study because of the possibility of embolization. They prefer carbon dioxide in their contrast studies of the urinary bladder. The radiopaque material, either aqueous dionosil or SH 167 L (Schering), provides a thin and satisfactory coating of the bladder mucosa and any lesion within the bladder.

After catheterization of the urinary bladder, 5–10 cc. of the radiopaque material is instilled. The amount of carbon dioxide, 100–200 cc., depends on the tolerance of the patient, usually when urgency is experienced.

Several exposures are made from various directions with the patient recumbent as well as erect. In selected cases, cineradiography is performed during micturition. By this method, better details were obtained concerning bladder diverticula and a variety of neoplasms.

Pelvic angiography was also used for information about extravesicular involvement in malignant lesions of the urinary bladder.—*J. Zausner, M.D.*

DESGREZ, H., HEITZ, F., VASSELLE, B., POROT, J., and GUYARD, G. Uréthrographie rétrograde par sonde souple à ballonnet. (Retrograde urethrography by flexible sound with balloon.) *J. de radiol., d'électrol. et de méd. nucléaire*, June-July, 1964, *45*, 306–308. (From: Service Central d'Electroradiologie de l'Hôpital de la Pitié, Paris, France.)

For urethrography the authors use a rubber sound 35 cm. long with a latex balloon at its tip. It does not differ from a retention catheter except for the smaller size of the balloon. A selection of sizes is kept available, usually No. 40, 28 and 12.

The sound is introduced a few centimeters and the balloon distended with water; a Cunningham clamp is placed at the base of the glans penis and then gentle traction assures occlusion of the urethra. Ten to 12 cc. of a water-soluble opaque medium is injected, and sagittal and both oblique view roentgenograms are made.

Xylocaine (1 per cent) may be used if needed. Octometine may be considered as an antispasmotic; this drug, however, is incompatible with water-soluble opaque media and thus it is employed only with lipiodol.—*Frank A. Riebel, M.D.*

SKELETAL SYSTEM

BONTE, J., DURSIN, C., CLAESSINS, H., and VAN DE VELDE, E. L'ostéolyse après contusion. Trois cas d'ostéolyse de l'extrémité acromiale de la calvicule. (Osteolysis after contusion. Three cases of osteolysis of the acromial end of the clavicle.) *J. belge de radiol.*, 1964, 47, 107–112. (Address: Dr. Bonte, Akademisch Ziekenhuis, De Pintelaan, Gent, Belgium.)

Post-traumatic osteolysis is a little known entity which has been described as occurring after fractures of the cranial vault, os calcis, and the necks of humerus and femur, as well as in certain dislocations. It is rare after simple contusion, but has been reported as occurring in the hip, pelvis, foot and especially in the clavicle.

The authors describe 3 cases in which contusions of varying degrees of severity had been sustained in the shoulder. Two of these were roentgenographed promptly and found to be negative. All 3 were again subjected to roentgenographic examination months later because of continued pain; at this time, osteolysis of the acromial ends of the clavicles was revealed in all 3.

In this entity the affected area of bone is completely absorbed, distinguishing it from post-traumatic necrosis of an epiphysis, which fragments and presents zones of irregular condensation. The differential diagnosis includes secondary osseous tumor, essential osteolysis without trauma, familial osteolysis and neurotrophic diseases.

The pathogenesis is obscure; various authors have attributed it to post-traumatic obliteration of the arterial supply, hypervascularization comparable to Sudeck's atrophy, or a neurotrophic factor.—*Frank A. Riebel, M.D.*

JONES, WALTER C., III, and MILLER, WALLACE E. Skeletal tuberculosis—1963. *South. M. J.*, Aug., 1964, *57*, 964–971. (From: Division of

Orthopaedics, University of Miami School of Medicine and the Jackson Memorial Hospital, Miami, Fla.)

In the elderly patient, bone and joint tuberculosis is manifested as an insidious low-grade disease and care should be taken to exclude it in cases with symptoms of arthritis.

The authors observed 36 patients, ranging in age from 3 to 82 years with a peak incidence between 30 and 50 years; 13 patients were Caucasian and 23 Negro.

There has been a sharp decline in the death rate from bone and joint tuberculosis in the U. S. In 1950, 44 per cent of the deaths were reported in patients over 55 years, whereas in 1959, 73 per cent of the patients were over 55 years.

The most frequent site of involvement was the spine and next the hip and knee. Three patients had no significant roentgenographic changes.

The authors briefly report the history of 5 cases with the pertinent roentgenograms. They recommend biopsy with smear, culture and guinea pig inoculation.

Treatment includes immobilization, specific drug therapy and pyridoxine. In adults in early cases, surgery is considered if the lesion can be excised whereas in children conservative therapy is indicated. In the late destructive stage, operation should be done as soon as possible—drainage, curettage, synovectomy or amputation.—*Martha Mottram, M.D.*

Rosenberg, Norman J. Osteochondral fractures of the lateral femoral condyle. *J. Bone & Joint Surg.*, July, 1964, *46-A*, 1013–1026. (From: Mt. Sinai Hospital, Cleveland, Ohio.)

The author reviews 15 cases of osteochondral fractures of the lateral femoral condyle. All of the patients were adolescents. The history was that of a twisting injury while the knee was flexed. A painful snap was heard and felt although the force was rarely violent. The joint filled with blood rapidly. Fat droplets were present in the joint effusion unless the fragment was undisplaced. Roentgenograms were obtained in the anteroposterior and lateral projections as well as in tunnel views.

In most cases, a small free fragment was apparent in the joint space. In others, a depressed defect was observed without a free fragment. In 3 incidences the roentgenograms were considered originally as normal.

Thirteen of the 15 patients had surgical treatment. In those with early exploration of the joint, normal hyaline cartilage was found with necrotic cancellous bone. Later exploration showed complete resorption of the bone but with viable cartilage remaining. This could not be differentiated from osteochondritis dissecans.

The author stresses that osteochondral fractures of the lateral femoral condyle are usually produced by forces transmitted through the patella but may occur without frank dislocation of the patella. He postulates that osteochondritis dissecans may in some cases be a sequela of minor trauma and urges prompt surgical exploration of acute injuries of the knee.—*Barry E. Gerald, M.D.*

Greville, Nicholas R. (Gainesville, Fla.) Osteochondritis dissecans: treatment by bone grafting. *South. M. J.*, Aug., 1964, *57*, 886–893.

The natural course of osteochondritis dissecans is sequestration of subchondral bone, failure of revascularization and reunion, with resultant degeneration of articular cartilage and separation of a bone fragment. The important pathology is the residual defect in the articular surface of the joint.

Three approaches are used in the treatment of osteochondritis in the knee: (1) before the fragment has separated the knee may be immobilized, sometimes for extended periods; (2) the fragment may be drilled; (3) after the fragment has separated, it may either be removed or fixed in place with pins or bone pegs.

The author presents the results of bone grafting in 5 knees in 4 patients; an additional patient was immobilized in a cast without surgery and one other had fixation of the loose body by 2 Kirschner wires which were later removed. When there was no bone to support the cartilage, grafts were applied and bone pegs placed through the fragment and grafts for fixation and to provide a vascular supply. The author found that this procedure shortens the period of inactivity, induces a blood supply and supports the cartilaginous surface. One patient had to have reexploration because of pain and a bone peg was noted projecting slightly into the joint.

Even though the roentgenograms showed some residual irregularity, clinical symptoms were relieved or improved in all patients.—*Martha Mottram, M.D.*

Tapia, Jorge, Stearns, Genevieve, and Ponseti, Ignancio V. Vitamin-D resistant rickets: a long-term clinical study of eleven patients. *J. Bone & Joint Surg.*, July, 1964, *46-A*, 935–958. (From: Department of Orthopedic Surgery, College of Medicine, State University of Iowa, Iowa City, Iowa.)

The most common form of rickets at the present time is the type resistant to vitamin D but unassociated with the chemical findings of renal tubular defects. Hypophosphatemia is the chief chemical finding. Serum calcium is mildly decreased and hyperphosphaturia is present. The disease is often familial but may occur sporadically. Roentgenologically, all forms of metabolic rickets resemble infantile rickets.

The authors present a clinical study of 11 patients with the diagnosis of vitamin D resistant rickets treated and followed for 8 to 46 years. The roentgenographic findings were characteristic, with widening of the epiphyseal plate and fraying of the metaphyses. Bowing deformities were common in the lower extremities. Growth retardation was present in all cases.

These patients were treated with high doses of vitamin D. Hypervitaminosis D was avoided by careful determination of the 24 hour urinary calcium levels. The deformities of the lower legs improved somewhat but did not clear completely. Surgical correction was often necessary. If the vitamin D therapy was interrupted or became inadequate, the deformities often recurred. Early diagnosis and prompt therapy are essential to prevent the severe deformities and to maintain a more nearly normal rate of growth.—*Barry E. Gerald, M.D.*

Accomazzi, Franco, and Gaudino, Fulvio. Considerazioni sul quadro radiografico del morbo di Cooley nell'adulto. (Considerations on the radiographic aspect of Cooley's disease in the adult.) *Arch. degli Istituti Ospedalieri S. Corona*, Mar.-April, 1964, *29*, 198-208. (From: Divisione Radiologica F. Cova, Pio Istituto Santa Corona, Milan, Italy.)

A case of Cooley's disease observed in a woman 25 years of age is reported.

The typical roentgen findings were noted particularly in the component bones of the vault of the skull, the ribs, clavicles, scapulae, left humerus, vertebrae and pelvic bones.

The roentgenologic diagnosis was confirmed by serologic studies, by the homozygotism of the patient (Rh−) and by the microcythemia in her parents' blood.

In the opinion of the authors, this is one of the few proven cases of Cooley's disease observed in an adult. —*A.F. Govoni, M.D.*

Blumenfeld, J. Isidoro. (Buenos Aires, Argentina.) La artropatía diabética: contribución a su estudio. (Diabetic arthropathy: contribution to its study.) *Rev. ortop. y traumatol.* (Latino Americana), Mar., 1964, *9*, 49-62.

Five cases of diabetic arthropathy of the region of the ankle and foot are reported and the literature is reviewed. One patient had changes localized to the tarsal bone, whereas the others had changes in the metatarsals or metatarsals and phalanges. The process goes through a series of stages from that of degenerative change through the severe stage of disintegration of the bone and joint surfaces with the irregular bone and fragments appearing quite opaque on the roentgenogram. The appearance is similar to that of other neuropathic joint diseases.—*Charles M. Nice, Jr., M.D., Ph.D.*

Pitts, Neal C. Myositis ossificans as a complication of tetanus. *J.A.M.A.*, July 20, 1964, *189*, 237-239. (Address: 2500 N. State Street, Jackson, Miss. 39216.)

The author reports 2 patients who developed myositis ossificans while recovering from tetanus. In both cases the lesions were around the elbows. Initially there was some difficulty in distinguishing this syndrome from serum sickness.—*Arthur E. Childe, M.D.*

BLOOD AND LYMPH SYSTEM

Leger, Lucien, and Mouktar, Marcel. Angiomégalies portales. (Portal angiomegalies.) *Presse méd.*, June, 1964, *72*, 1859-1864. (From: Clinique Chirurgicale de l'Hôpital Cochin, Paris, France.)

Venous dilatations of the portal system may develop either as diffuse or localized angiomegalies. The causes fall into three classes: (1) augmentation of intravascular pressure, either from the passive effect of portal obstruction (cirrhosis, thromboses, compressions) or the active effect of the opening of splenic, gastric or mesenteric shunts; (2) increase in the speed of circulation which is not necessarily correlated with hypertension, as from the influx of arterial blood via peripheral shunts, although the liver is normal; and (3) dilatable character of the veins themselves and their surrounding tissues. This is especially apt to occur in children.

In the diffuse angiomegalies the portal vein is usually verticalized, with the surcharged splenic vein joining it at a nearly right angle; this is unfavorable and possibly explains the sinuosity of the splenic vessel. The calibers of vessels which serve the classic refluxes (gastric and perisplenic veins) are relatively modest. These diffuse angiomegalies are of long standing, and are nearly always associated with marked splenomegaly.

Localized angiomegalies may be considered as functionally adequate porto-caval shunts if they prevent the formation of esophageal varices, if splenomanometric readings are normal, and if preoperative clamping produces a rise in portal pressure. This requires a vessel with a caliber of about 15 mm. Much more rarely a large capillary bed can function as a fairly adequate shunt, but with hypertension. These are cases in which monstruous ectasias have developed over a long period; they act as reservoirs which alternately fill under the blows of hypertension from the splenic and mesenteric arteries, they slowly redistribute blood toward the portal system and in some cases toward the caval system as well as across many small anastomoses.—*Frank A. Riebel, M.D.*

Baird, R. J., and Doran, M. L. The false aneurysm. *Canad. M. A. J.*, Aug. 8, 1964, *91*, 281–284. (From: University of Toronto Cardiovascular Unit of the Toronto General Hospital, Toronto, Ontario, Canada.)

The development of a false aneurysm is a serious complication and frequently leads to loss of the patient's limb or occasionally of his life. In this era, the commonest cause is disruption of vascular anastomoses following reconstructive arterial surgery. A second major cause is direct arterial trauma which results in disruption of the arterial wall. Of significance in this category are knife wounds, bullet wounds and, in recent years, needle punctures for such diagnostic procedures as cardiac catheterization, arterial pressure measurements, and angiography. The lesion is particularly prone to occur following needle punctures in patients with aortic insufficiency. The wide pulse pressure prevents sealing of the arterial defect, and perpetuates the opening into the hematoma cavity.

The classic false aneurysm presents clinically as a pulsating mass with a systolic bruit. It expands slowly, causing increasing pain and tenderness until it ruptures. There may be associated infection, with oozing of pus or blood. In 4 of the 18 patients with a total of 25 false aneurysms, the aneurysm was originally diagnosed as an abscess. In 1 of these the lesion was lanced in a physician's office, with catastrophic results. Other lesions which may require differentiation include arteriovenous fistulae, true aneurysmal dilatation of an artery, normal pulsation transmitted through a mass of glands or tumor or abscess, or a pulsating bursa, *e.g.*, in the popliteal fossa.

The treatment is surgical excision with restoration of arterial continuity. Continuity may be restored by oversewing the defect or inserting a patch graft, or replacing a segment with a new arterial substitute.—*Douglas S. Kellogg, M.D.*

Winckelmann, G., Hiemeyer, V., Weissleder, H., and Schoop, W. Thrombolytic treatment of acute arterial occlusion with streptokinase. *German Med. Monthly*, June, 1964, *9*, 249–252. (From: Department of Medicine, University of Freiburg, Freiburg, Germany.)

The authors treated 19 patients with acute peripheral arterial occlusions by continuous infusions of highly purified streptokinase. There were 6 cases of embolism, 9 of thrombosis and 4 of acute arterial occlusion of uncertain origin. No serious side reactions were observed despite relatively rapid injection of the initial dose and prolonged high dose infusion. In 9 cases all the peripheral arterial pulses returned, in 5 there was improvement and in 5 there was no improvement. Arteriography carried out in 9 patients showed results which were in close agreement with the clinical findings.

Better results were obtained with emboli than with thrombi. Arteriographic observations showed that thrombotic occlusions were usually much longer than embolic occlusions and that the entire length of a thrombus could not be resolved.—*Arthur E. Childe, M.D.*

Calnan, J. S., Kountz, S., Pentecost, B. L., Shillingford, J. P., and Steiner, R. E. Venous obstruction in the aetiology of lymphoedema praecox. *Brit. M. J.*, July 25, 1964, *2*, 221–226. (From: Department of Surgery and Radiology and the M.R.C. Cardiovascular Research Group, Postgraduate Medical School, and Hammersmith Hospital, London, England.)

Lymphoedema praecox is defined as idiopathic edema appearing before the age of 35 years. The process is usually unilateral and affects women predominantly. The onset in the majority of cases is between the ages of 9 and 25 years.

The authors studied the venous drainage in 23 consecutive patients with lymphoedema praecox. The venous drainage from the affected limb was investigated by percutaneous femoral venography. The venous flow and pressure measurements were obtained in the common iliac veins. Lymphangiography was successfully performed in 10 cases.

In 11 patients, venous obstruction was demonstrated at the ileo-caval junction. This was shown as a compressing band with decreased contrast density at the point where the right common iliac artery crosses the left common iliac vein. The vein was widened at the point of obstruction with mild proximal dilatation of the immediately adjacent portion. Collateral vessels were often filled. Using a horizontal beam with the patient in the supine position, venous obstruction at the ileo-caval junction was marked. Although flexion of the hips decreased the compression somewhat, it was not completely relieved in any case. The flow and pressure studies in the 11 patients with venous obstruction showed decreased flow in 7 patients, each of whom exhibited roentgenographically a complete block of the left common iliac vein. In the remaining 4 patients flow studies were normal. The obstruction did not appear complete in the roentgenologic studies but was made so by dorsi-flexion at the lumbosacral junction.

Successful lymphangiograms were performed in 6 of the 11 patients with venous obstruction. Three were normal. The other 3 showed hypoplastic lymphatics on the affected side. Successful lymphangiograms were obtained on 4 patients without venous obstructions; 2 showed hypoplastic lymphatics and 2 normal lymphatic channels.

From these data, the authors postulate that primary lymphedema in the left lower extremities is related to venous obstruction at the ileo-caval level. This obstruction may be due to a compression anteriorly by the right common iliac artery associated with posterior compression against the 4th lumbar vertebra. The posterior compression is more marked in women because of the increased lumbar lordosis in the female. It has been shown that a raised venous pressure can impede both the flow and reabsorption of lymph. The combination of lymphatic inadequacy and an increase in venous pressure may be sufficient to produce lymphedema. It appears unlikely that lymphedema is secondary to changes in lymphatics only. The mechanism suggested by the authors would apply only to the left leg. Further studies of the venous pressures in patients with lymphedema while supine and standing is suggested as well as comparison with a group of controls.—*Barry E. Gerald, M.D.*

Tilak, S. P., and Howard, John M. The influence of the dual circulation on the viability of lymph nodes following interruption of their blood or lymphatic supply. *Surg., Gynec. & Obst.*, Aug., 1964, *119*, 349–352. (From: Department of Surgery, Hahnemann Medical College, Philadelphia, Pa.)

The dual circulation of the liver by the hepatic artery and portal vein and of the lung by the pulmonary and bronchial arteries has been well known. The fact that the lymph nodes have a dual circulation has also been well reocgnized but in a different context. The purpose of this investigation has been to study the role of the two circulations of the lymph node; *i.e.*, arterial and lymphatic, as to their respective ability to sustain the viability of the lymph node. Should either circulation suffice to maintain supply of the lymph node, autografting of lymph nodes into the bed of a radical lymph node resection might become feasible by the restoration of a single circulatory pathway.

In order to study the survival of the lymph node after interference with its circulation two sets of operations were performed: (1) division of the blood vessels—artery and vein—to a lymph node, preserving intact all the lymphatics; 9 dogs were studied in this series; (2) division of all lymphatics—afferent and efferent—to a lymph node, without interfering with its arterial and venous supply; 10 dogs were studied in this series.

The popliteal area of the dogs was used in these studies as there was usually a single lymph node in this region. Subcutaneous lymphangiography was carried out before the operation by injection of the opaque medium into the plantar surface of the distal half of the paw at 2 or 3 sites. The area of injection was thoroughly massaged and firm pressure was maintained over the paw. The limb was passively moved at the hip, knee and ankle joints through their full range. Lateral roentgenograms of the limb were then taken, 1 or 2 minutes after injection, which showed the popliteal lymph node with its afferent and efferent lymphatics.

Following preliminary lymphangiography, the dogs were operated upon. The popliteal lymph node, embedded in subcutaneous fat, was easily found. After partial exposure of the lymph node, Evans blue dye was injected into the paw thereby permitting more accurate visualization of the lymphatic vessels at the level of the lymph node. Subsequent to this visualization, the lymphatics or the blood vessels were divided.

Subcutaneous lymphangiography was repeated on the respective limbs postoperatively, weekly during the first month, every 2 weeks during the second month, and then once a month for a total follow-up period of 5 months. The lymph node wsa removed for histologic examination at the end of the fifth month.

The authors conclude that the popliteal lymph node of the dog may survive *in situ* if either the blood flow or the lymph flow is maintained. Interruption of the blood supply is followed by the maintenance of lymphatic circulation through the lymph node and rapid restoration of a normal roentgenographic lymph nodes pattern. The division of the afferent and efferent lymphatic vessels to the lymph node is usually followed by restoration of the lymphatic circulation through the lymph node within a period of some 2 to 3 weeks.—*C. Peter Troug, M.D.*

GENERAL

Capotorti, L., Ferrante, E., and Bottini, E. Sindrome di De Toni-Debré-Fanconi senza apparente cistinosi in un bambino di due anni. (De Toni-Debré-Fanconi syndrome without demonstrable cystinosis in a 2 year old child.) *Riv. clin. pediat.*, Nov., 1963, *72*, 318–338. (From: Clinica Pediatrica dell' Università di Roma, Rome, Italy.)

A case of De Toni-Debré-Fanconi syndrome with demonstrable cystinosis in a child, age 2 years and 3 months, is described. The picture of the syndrome was complete: delay in growth, severe rickets, glycosuria of renal type, hyperaminoaciduria, hypophosphatemia, considerable increase of alkaline phosphatase in the serum and hyperchloremic metabolic acidosis. Relative hyperphosphaturia and hypercalciuria were also found.

On reviewing the literature, the authors concluded that in the De Toni-Debré-Fanconi syndrome we have a combination of an abnormal intestinal and tubular absorption of phosphorus and that both together help to maintain the low phosphatemic level and determine the particular resistance to therapy.

With the doses used in their case (30,000 IU of vitamin D pro die), the authors observed a hypercalciuria without deposit of calcium within the skeleton (the reverse of what occurred in rickets secondary to vitamin deficiency). Evidently, with these doses the intestinal absorption of calcium is improved, but not its conversion within the organism through the other two effector organs of the calcium metabolism: the skeleton and the kidney. The resistance of rickets to normal prophylactic and therapeutic doses of vitamin D may be attributable, on the basis of recent studies on vitamin D activity of the serum (Ca^{45}-AVDS), to a lack of response of the target organs (intestinal cells—cartilaginous cells—epithelial cells).

The alkalines by themselves were ineffective on rickets and acidosis. The metabolic acidosis can be explained by an excessive acid production (ketosis secondary to a depletion of the hepatic glycogen storage as an effect of the glycosuria) and also by an abnormal tubular absorption of the bicarbonate (urinary loss of fixed basis).

The authors explain the hyperchloremic acidosis observed in their cases as a form of compensation to the diminution of blood ions (HCO_3) necessary for the maintenance of the ionic equilibrium (an increase of chloremia, perhaps induced with NH_4Cl or $CaCl_2$, is followed by a reduction of bicarbonate in the blood); therefore, the organism responds with an increase of the chloremia every time that the bicarbonates decrease.

The prognosis of the De Toni-Debré-Fanconi syndrome, so-called idiopathic (nonsecondary to a tubular lesion due to accumulation of cystine), is generally more benign.

The authors think that the resistance of the De Toni-Debré-Fanconi syndrome could perhaps be surmounted with high doses of vitamin D, varying according to the cases under careful continuous control of the calcemia to avoid hypervitamin intoxication in immobilized patients.

The genetic problem is still unresolved. However, it seems that the De Toni-Debré-Fanconi syndrome with cystinosis (probably recessive autosomic in type) could be genetically separated from the type so-called idiopathic or cryptogenetic without cystinosis.—*Cesare Mazzucco, M.D.*

Moran, John M. An unusual foreign body. *J.A.M.A.*, Aug. 10, 1964, *189*, 511–512. (Address: 303 North Street, Hingham, Mass.)

It is noted that occasionally the persistence of a small force can make itself felt in a large way. A 68 year old white male was admitted to the Boston City Hospital complaining of painful ulcerations of the left leg. These had been present for about 2 weeks. Bilateral leg varicosities had been present for more than 20 years. He had periodically dressed the ulcers with rags. He had had no diabetes, nor any previous leg ulcerations, venous surgery, or thrombophlebitis. On the left leg a circumferential purulent ulceration extended from the level of the malleoli 14 cm. cephalad, with cellulitis to the knee. There were prominent saphenous and lesser saphenous varicose veins bilaterally. All pulses were present. Roentgenographic examination of the leg revealed no osteomyelitis.

Treatment included bed rest with leg elevation, moist packs and antibiotics; improvement was prompt. However, within 2 weeks multiple draining sinuses began to appear just above the malleoli. It became evident at this time that dorsiflexion of the left foot was not possible and that plantar flexion was weak. Roentgenograms showed a punched out lesion on the lateral aspect of the supramalleolar fibula, with abundant callus formation nearly bridging across the defect.

Because of persistent drainage from multiple sinus tracts, operative debridement was done. In the depths of the circumferential wound was found an ordinary rubber band which had eroded through the anterior tibial tendon, and into the lateral aspect of the fibula. This was a wide rubber band. It was removed and a liberal debridement was completed. It was evident by testing that the superficial peroneal and sural nerves had also been compromised by the rubber band.

On questioning, it was learned that the patient had kept his rag dressing in place with rubber bands. After skin grafting complete healing was obtained.—*Douglas S. Kellogg, M.D.*

Burrow, Gerard N., Hamilton, Howard B., and Hrubec, Zdenek. Study of adolescents exposed *in utero* to the atomic bomb, Nagasaki, Japan. *Yale J. Biol. & Med.*, June, 1964, *36*, 430–444. (From: Departments of Medicine, Clinical Laboratories, and Statistics of the Atomic Bomb Casualty Commission, Hiroshima and Nagasaki, Japan.)

With the increasing use of atomic energy in our society, the effects of ionizing radiation, particularly those that are delayed, have become a matter of great concern. Since 1947 the Atomic Bomb Casualty Commission has conducted continuing medical studies among survivors of the atomic bombing in Hiroshima and Nagasaki. Since 1951 this has included a group of children who were irradiated *in utero*. Fetal tissues being particularly sensitive to ionizing radiation, this latter group is of special interest.

An intensive 2-year study of 286 Nagasaki adolescents, ages 13 to 14, was conducted as part of a long term program to determine possible differences in growth and development which might be attributed to "*in utero*" exposure to the atomic bomb. Three comparison groups were studied: Group I, whose mothers were within 2,000 meters of the hypocenter; Group II, whose mothers were located between 3,000 and 5,000 meters from the hypocenter; and Group III, whose mothers were not in the city at the time of the bombing.

Mental retardation was found with significantly higher frequency in Group 1 males, but not in the females. The explanation for this difference is not known. Females in Group 1 received on the average a somewhat higher dose than the males, so it is unlikely that radiation dosage is the explanation. Group 1 females who were in the first trimester of gestation at the time of the bombing, and whose mothers experienced the acute radiation syndrome, had poorer visual acuity than comparable children of asymptomatic mothers. However, the small number of observations requires caution in interpretation of this finding.

No cases of leukemia or malignancy were found, but the sample was not large enough to detect even a tenfold increase in incidence. No significant differences attributable to a radiation effect were noted among the comparison groups in regard to radial pulse, blood pressure, hematologic, or urine examinations. Parasitic infestation occurred in about one-third of the subjects, and Group 1 had a significantly higher incidence of infestation than did the other comparison groups. Trichocephalus and Ascaris infestation accounted for the higher prevalence. Hookworm occurred less frequently. It is suggested that these findings are probably associated with different dietary and living habits.—*Douglas S. Kellogg, M.D.*

RADIATION THERAPY

BREED, J. ERNEST. Radium therapy in cancer of the eyelid. *IMJ, Illinois Med. J.*, 1964, *125*, 237–241. (Address: Associate in Radiology, Northwestern University Medical School, Chicago, Ill.)

Between 1934 and 1961 the author had treated 209 cases of cancer of the eyelid, virtually all with radium.

The beta rays of radium were the chief form of therapy in 167 patients. A quarter strength beta plaque screened with 0.1 mm. Pb was applied, extending 5 mm. beyond the margin of the tumor. A total of 7 to 8 hours treatment time divided in 6 to 9 treatments was given over a 2 to 3 week period.

If the lesion was over 6 mm. thick, gamma radiation was used. The eye was shielded with a gold plate and an applicator containing 50 to 200 mg. of radium was placed at a distance roughly equal to the thickness of the tumor. Twelve to 15 treatments of 15 to 20 minutes each were given over a 2 to 3 week period. The amount of radium in the applicator varied with the source skin distance.

Of the total, 103 patients were living and well 3 years after treatment, representing 87 per cent of the determinate cases. Ninety two were indeterminate, largely due to loss to followup.

No cataracts were detected.—*Henry J. Klos, M.D.*

LEE, E. STANLEY, and MACKENZIE, D. H. Osteosarcoma: a study of the value of preoperative megavoltage radiotherapy. *Brit. J. Surg.*, April, 1964, *51*, 252–274. (Address: E. Stanley Lee, Consultant Surgeon, Westminster Hospital, London, England.)

In 1951, at the Westminster Hospital, an experiment was started, consisting of preoperative irradiation of osteosarcoma followed by elective ablation of the tumor. The following is the authors' experience in 157 consecutive cases of previously untreated osteosarcoma without metastases.

The program called for irradiation of the tumor with 2 mev. roentgen rays at a rate of 1,000 r tumor dose weekly to a total of 7,000–8,000 r. Surgical removal was then delayed as long as clinical improvement was noted, often up to 3 or 4 months. About half the time removal of the tumor was not performed chiefly because metastases had occurred since the beginning of therapy but often also because of an unsuitable site or refusal of amputation.

Using the group of 92 patients eligible for evaluation of a 5 year survival rate, there were 20 alive without recurrence—a cure rate of 21.8 per cent, which is higher than in most published series. Those patients with tumors involving the femur had a 5 year survival rate of 30 per cent.

The superior results would imply that the delay before amputation caused no harm and that the patients who developed discernible metastases after beginning therapy had had undiscernible metastases from the beginning and were spared a mutilating procedure.—*Henry J. Klos, M.D.*

RADIOISOTOPES

SARGENT, THORNTON, LINFOOT, JOHN A., STAUFFER, HENRY, and LAWRENCE, JOHN H. Use of a whole body counter in turnover studies with Ca 47. *J. Nuclear Med.*, June, 1964, *5*, 407–416. (From: Donner Laboratory of Medical Physics and Biophysics, University of California, Berkeley, Calif.)

Previous investigations of calcium metabolism by workers using Ca^{47} and Sr^{85} have been incomplete because they failed to take into account the component of serum calcium disappearance. The authors suggest that the whole body counter can be used to advantage because it can measure the calcium deposit in bone without relying on calculation from serum measurements.

They studied a group of 14 patients using the Argonne type whole body counter. Ca^{47} was injected intravenously in a dose of 2 to 25 μc. A count of the patient 5 minutes following injection was considered 100 per cent and subsequent measurements were made daily for several days and then at 7 day intervals. Urine, blood, and stool sample determinations were also made and counted in a standard well counter with pulse height discrimination against the Sc^{47} daughter of Ca^{47} and against I^{131}.

The first group of 4 patients were those with normal bone metabolism. These all revealed retention curves of similar nature, with an initial half-time of 28 to 30 days.

The second group consisted of 4 female patients with mammary carcinoma and osseous metastases. They showed greatly increased turnover rates of calcium. Those who received radiation therapy showed a normal retention curve following the course of treatment.

The final group of 6 patients were those with acromegaly. It was found that this group deposited far more calcium into the bone compartments than normals. The blood, urine, and fecal specific activity curves were parallel in most cases. Serum turnover was plotted and compared with whole body retention data. It was noted that the findings did not correlate with the clinical severity of the disease. For this reason, the authors have proposed to determine the degree of activity of acromegaly and the effect of therapy by whole body counting. Future studies are planned with Ca^{47} and Sr^{85} simultaneously in order to extend the retention curves beyond the relatively short time possible with Ca^{47} alone.—*Millard N. Croll, M.D.*

Rosenthall, Leonard. The use of strontium-85 for the detection of bone lesions. *J. Canad. A. Radiologists*, June, 1964, *15*, 53–59. (From: Montreal General Hospital, Montreal, Quebec, Canada.)

This report deals with the clinical evaluation of strontium 85 in over 200 cases during a 12 month period.

Sr^{85} has a physical half life of 65 days and emits a single gamma photon of 0.510 mev. The author administered the isotope as strontium chloride by intravenous or intramuscular routes, giving a dose of 75–100 μc when automatic scintillation scanning was used; and a dose of only 30–50 μc when external spot counting was employed. It has been estimated that the total body dose is 2.3 rads per 100 μc injected. Counting is performed at least 1 day and no later than 7 days after the injection. In any routine survey for metastatic disease, counts are made with an external counter over the seventh cervical vertebra and then distally at 10 cm. intervals. The activity over the ankles is also determined and serves as a control.

The author states that greater accuracy is obtained for the discovery of focal metastatic disease if the counts at the 10 cm. levels are compared to the standard taken over the seventh cervical vertebra. However, if there is diffuse metastatic involvement of the axial skeleton, a comparison with the standard taken over the ankles is more valid.

The ability of Sr^{85} to demonstrate traumatic fractures of the base of the skull is discussed. Two cases are presented to substantiate this claim.

The author points out that the concentration of strontium is a nonspecific finding and must be correlated with the clinical history and the roentgenologic appearance of the region under scrutiny. In the 200 cases presented, Sr^{85} uptake was observed in pyogenic osteomyelitis, tuberculosis, fibrous dysplasia, degenerative and rheumatoid arthritis, hyperostosis frontalis interna, Paget's disease, chronic sinusitis associated with osteitis, growing epiphyses and surgical osteotomy. The concentration of Sr^{85} in the presence of Paget's disease, growing epiphyses or degenerative arthritis may tend to give false positive counts or even obscure the presence of a primary or secondary tumor in the area. Sr^{85} is excreted in the stool and again may give high pelvic counts because of this for as long as 8 days after injection.

It is apparent that this technique cannot differentiate benign from malignant tumors, or between the various non-neoplastic conditions encountered. Its chief value lies in its ability to uncover bony lesions before they are visible on roentgenograms. This, if for no other reason, establishes the value of the study.—*Kenneth M. Nowicki, M.D.*

DiGiulio, Walter, and Beierwaltes, William H. Parathyroid scanning with selenium 75 labelled methionine. *J. Nuclear Med.*, June, 1964, *5*, 417–427. (From: Departments of Internal Medicine [Nuclear Medicine], University of Michigan Medical School and United States Veterans Administration Hospital Radioisotope Service, Ann Arbor, Mich.)

Visualization of the parathyroid gland *in vivo* by photoscanning was suggested by the authors in 1962 using Co^{57} B_{12}. It was then demonstrated by Potchen that tritiated methionine concentrated in rat parathyroids, and this paper presents work on selenium 75 methionine studies in humans and dogs.

The authors scanned 3 patients with hyperparathyroidism, 2 cases being associated with confirmed hyperfunctioning parathyroid adenomas. The patients were prepared with a low calcium, high protein diet. A dose of 300 μg. of triiodothyronine was administered daily for 5 days and followed by 250 μc of Se^{75} methionine intravenously. In 1 patient the parathyroid adenoma was visualized by scanning 8 hours after injection. A second patient had only suggestive concentration and the third patient showed no evidence of scanning localization.

The authors then studied the relative tissue concentrations of the radioisotope in the first 2 patients and found that the parathyroid contained 2 to 3 times the activity present in the peripheral blood, thyroid gland, or muscle. The radioactivity concentrations in the blood following the injection of Se^{75} methionine in dogs was found to be identical to the curves previously reported by Oldendorf in humans. The lowest portion of the blood curve occurs at 30 minutes rising to a plateau at 6 hours. An experimental phantom was built with the target/non-target ratio based on the results of the tissue concentration study. It was seen that at these levels of concentration the mock tumors were easily detected by scanning.

Preliminary dosimetry studies suggested a biologic half-life of the Se^{75} of 23 to 36 days. This would yield a whole body radiation dose of about 615 mrads. The authors indicate, however, that this may need revision after completion of long term studies, which are now being conducted. Electrophoretic patterns reveal that the injected Se^{75} methionine, after diminishing in the blood, appears uniformly distributed in the serum proteins. It is apparently incorporated in both protein of the plasma and in the red blood cells, but disappears more rapidly with time from the plasma.

Ten dogs were then studied to evaluate the effect of the various parameters of preparation prior to the administration of the Se^{75} methionine. No apparent effect on the uptake of the various tissues was seen as a result of altering these parameters.

The authors conclude that two sources of variability in the photoscanning results may be due to (1) impurities of the Se^{75} methionine causing limitation of maximum rate of uptake, and (2) uptake of the Se^{75} methionine by the bone marrow of the cervical spine and sternum.—*Millard N. Croll, M.D.*

CHEMOTHERAPY

GREENBERG, LOWELL H., and TANAKA, KOUICHI R. Congenital anomalies probably induced by cyclophosphamide. *J.A.M.A.*, May 4, 1964, *188*, 423–426. (Address: Dr. Tanaka, 1000 W. Carson Street, Torrance, Calif.)

A woman with Hodgkin's disease who was treated with cyclophosphamide during pregnancy was delivered of a child who showed multiple congenital anomalies. The patient received intravenous cyclophosphamide during the first trimester of pregnancy and then was placed on maintenance oral dosage of the drug throughout the remainder of the pregnancy. She also received roentgen therapy to the neck and mediastinum before the time of conception.

The congenital defects in the child were absence of the first toe and metatarsal bilaterally, hypoplastic middle phalanx of fifth finger, bilateral inguinal hernia, grooves in the hard palate, flattened nasal ridge and a skin tag on the anterior midabdomen. Chromosomal study was normal. The most intensive therapy was administered in the period corresponding to the seventh or eighth week of fetal life.

The authors feel that cyclophosphamide can reasonably be incriminated as a mutagenic agent in this case.—*James R. Knapp, M.D.*

SPEED, DOROTHY E., GALTON, D. A. G., and SWAN, A. Melphalan in the treatment of myelomatosis. *Brit. M. J.*, June 27, 1964, *1*, 1664–1669. (From: Chester Beatty Research Institute, Institute of Cancer Research, Fulham Road, London, England.)

The basic abnormality in myelomatosis is the progressive proliferation of abnormal plasma cells giving rise to the development of progressive destructive lesions of the skeleton which result in pain, progressive replacement of the bone marrow, hematopoietic insufficiency and long continued proteinuria and hypercalcuria with consecutive renal damage. Unfortunately, with the exception of radiation therapy, the available forms of treatment are not very effective.

The authors report their experience in the treatment of 20 patients with myelomatosis using melphalan, L-phenylalanine nitrogen mustard. One group of patients had been previously treated using roentgen therapy and other chemotherapeutic agents (13 patients with a median follow-up of 10 months); the second group received no previous therapy (7 patients with a median follow-up of 11 months). The follow-up for the entire group ranged from 6 to 33 months and the study was carried out from December, 1959 through September 1, 1962. Ten patients in a group were immobile because of pain, 5 were ambulatory but with pain, and 5 were essentially asymptomatic.

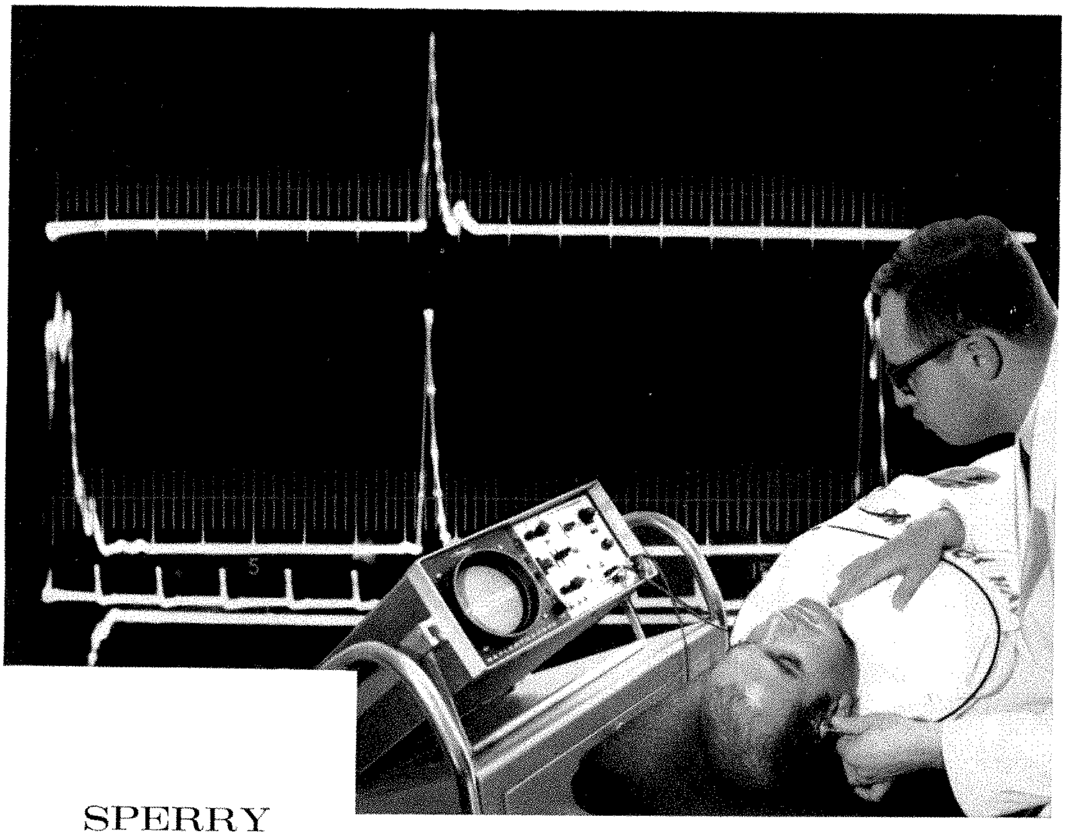
The choice of melphalan therapy was based on the following points: 1. widespread disease in which local roentgen therapy was not practical; 2. there had been no objective response to previous therapy; and 3. hematologic abnormalities such as anemia, purpura, ecchymoses.

The melphalan was administered orally in daily doses of 5 to 10 mg. for a total dose in each treatment session of 1 mg. per kilogram. Treatment was repeated at intervals if it had proved helpful and within the limits of bone marrow toxicity. Other ancillary therapies were blood, analgesics, prednisone, adrenocorticotrophin, methandienon, nandrolone, stilbestrol or local roentgen therapy. The major limiting factor for therapy was bone marrow toxicity manifested by a depressed hemoglobin, neutrophil count, and platelets. Uremic patients tolerated treatment poorly.

Pain relief was most striking in 10 of the 20 patients treated; 7 of the 10 bedridden patients were made ambulatory. The other results from therapy were weight gain and improvement in the laboratory finding such as hemoglobin, diminution in the number of plasma cells in the bone marrow, depression of the abnormal globulins, depression of the sedimentation rate, reduction of the blood urea nitrogen and diminution in excretion of urinary protein. Roentgenographic changes in bones were unaffected by the drug with no significant progress or healing of the bone lesions. Progress of the roentgenographic lesions was noted in 1 case.

The improvement from the medication was slow to appear and therefore treatment should be pursued over a long period of time. Death occurred in 7 of the 20 patients 1 to 14 months following treatment.

Melphalan therapy is a beneficial agent in the treatment of myelomatosis as demonstrated by the authors.—*Luther W. Brady, M.D.*



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2. A. Jefferson, Acta Neurochirurgica, x4(1962) 392-409
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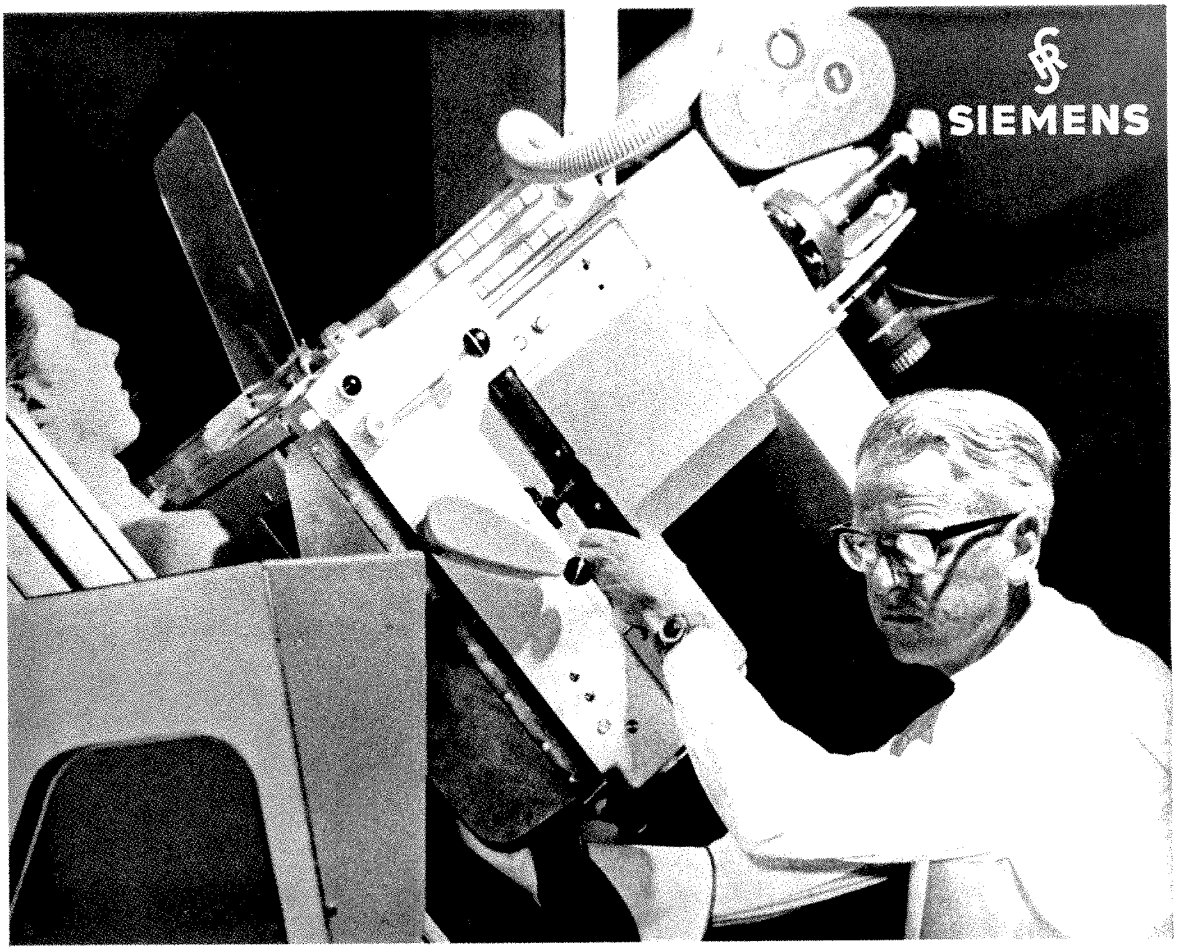

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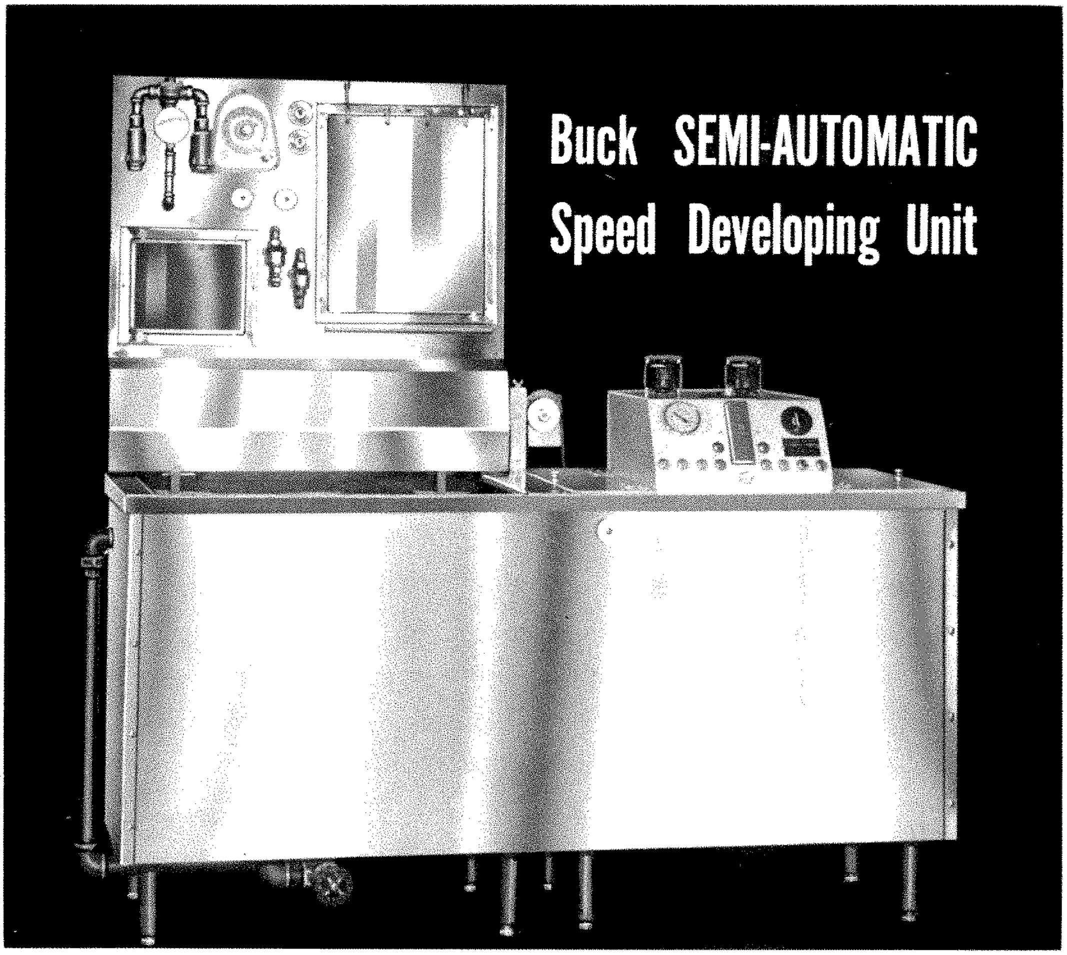

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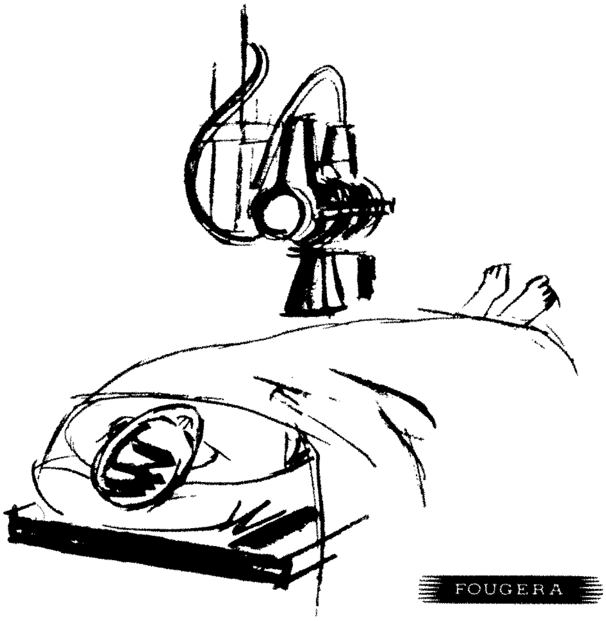

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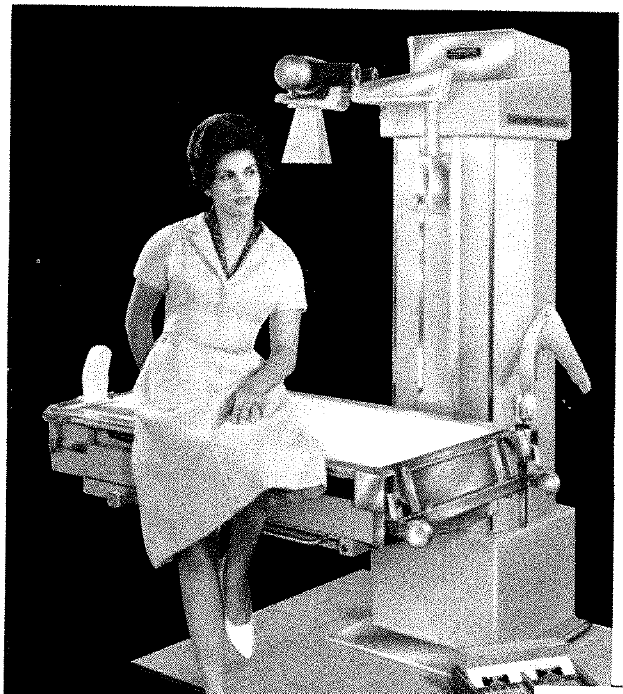

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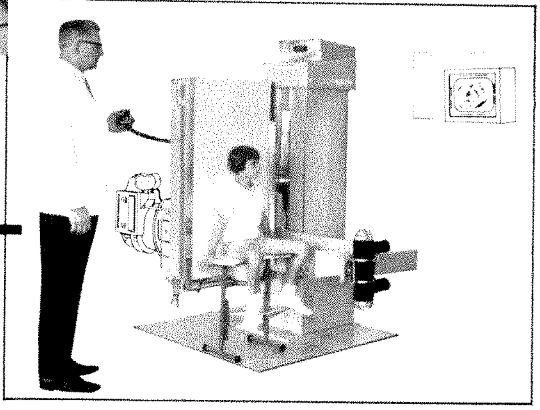

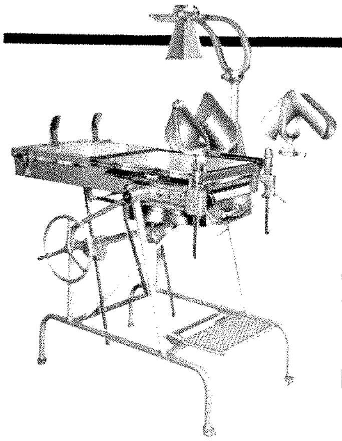

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INDEX TO ADVERTISERS

Aerojet Delft Corporation xxxii
Ansco (General Aniline & Film Corp.) xxx
Automation Industries (Sperry Division) xlvii

Barnes-Hind Barium Products liv
Bell-Craig, Inc. xviii
Buck X-Ograph Company xlix

Cenco X-Ray Corporation .... xvi, xvii, xxv

du Pont, E. I. de Nemours Co. .... x, xi

Eastman Kodak Company .... xlv, xlvi

Fougera, E. & Company, Inc. .... viii, l

General Electric Company .... vi, xxxiii, xxxiv, xxxv, xxxvi

Halsey X-Ray Products Company liii
High Voltage Engineering Corp. xliv
Hunt, Philip A., Chemical Corporation xxxi

Ilford Inc. xxvii

Keleket X-Ray Corporation .... xxviii, xxix

Liebel-Flarsheim Company (Division of Ritter Co.) li
Low X-Ray Corporation xv

Machlett Laboratories xix
Mallinckrodt Chemical Works .... xxi, xxii, xxiii, xxiv
McElroy, Donald, Inc. l

North American Philips Company .... xxxviii, xxxix
Nuclear-Chicago Corporation vii

Picker X-Ray Corporation .... ix, xliii, Fourth Cover
Profexray, Inc. .... i, xiii, xl, xli

Schick X-Ray Company xii
Siemens Medical of America, Inc. xlviii
Squibb, E. R., & Sons .... v, Third Cover
Standard X-Ray Company xx

Technical Measurement Corporation xiv
Thomas, Charles C, Publisher xlii

Viso Corporation xxvi

Westinghouse Electric Corporation .... Second Cover
Winthrop Laboratories xxxvii

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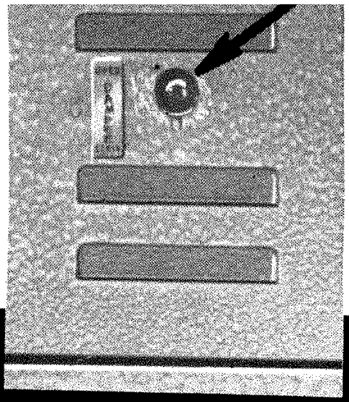

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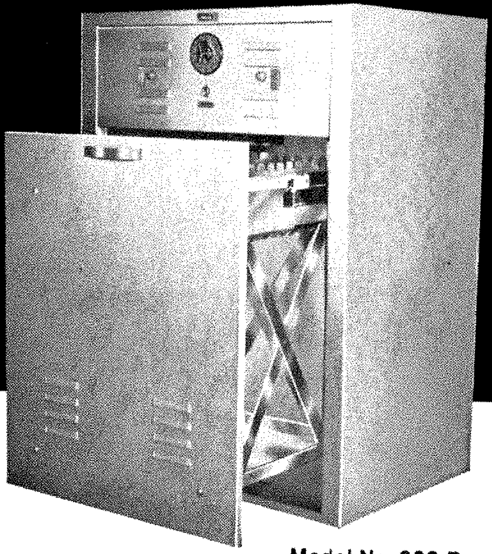

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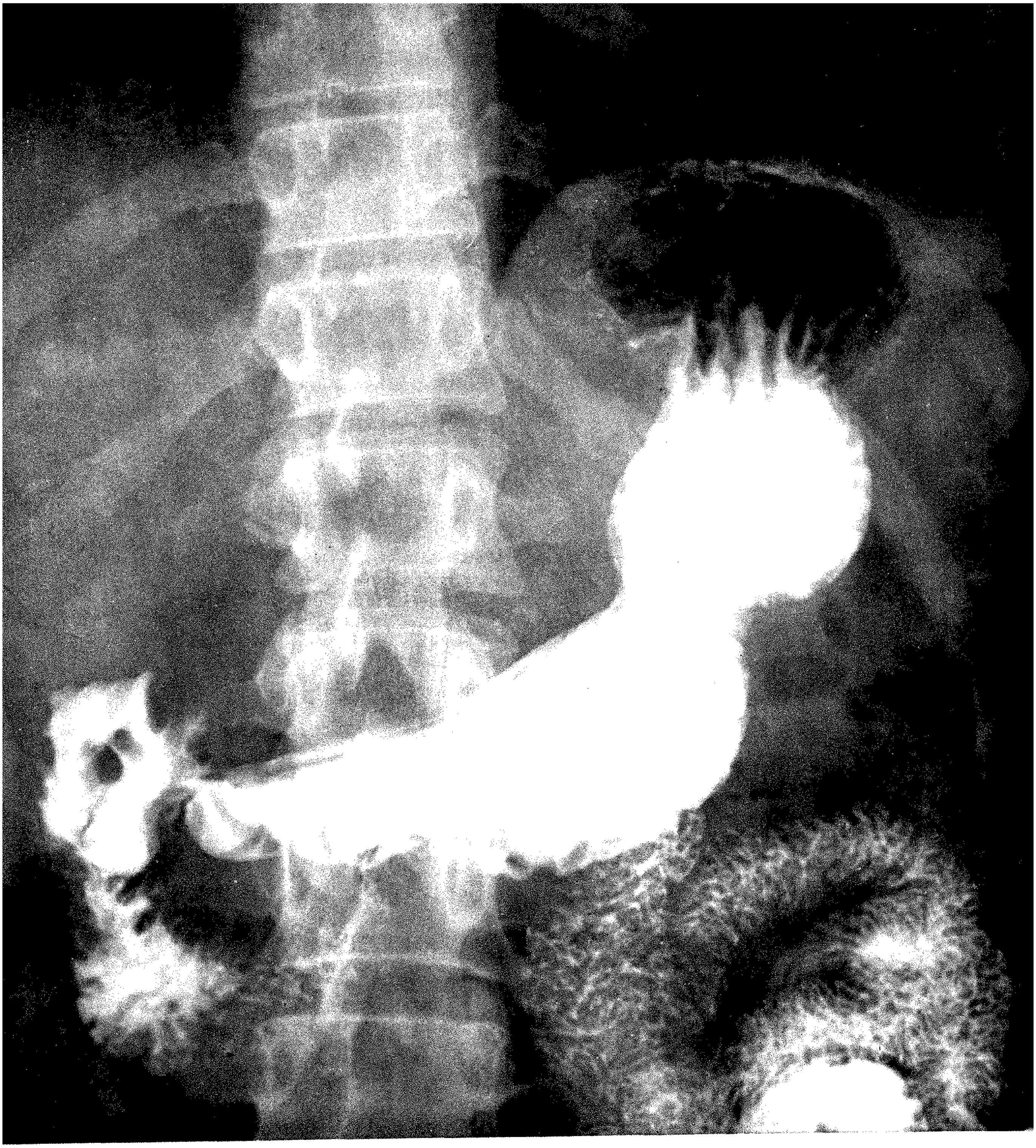

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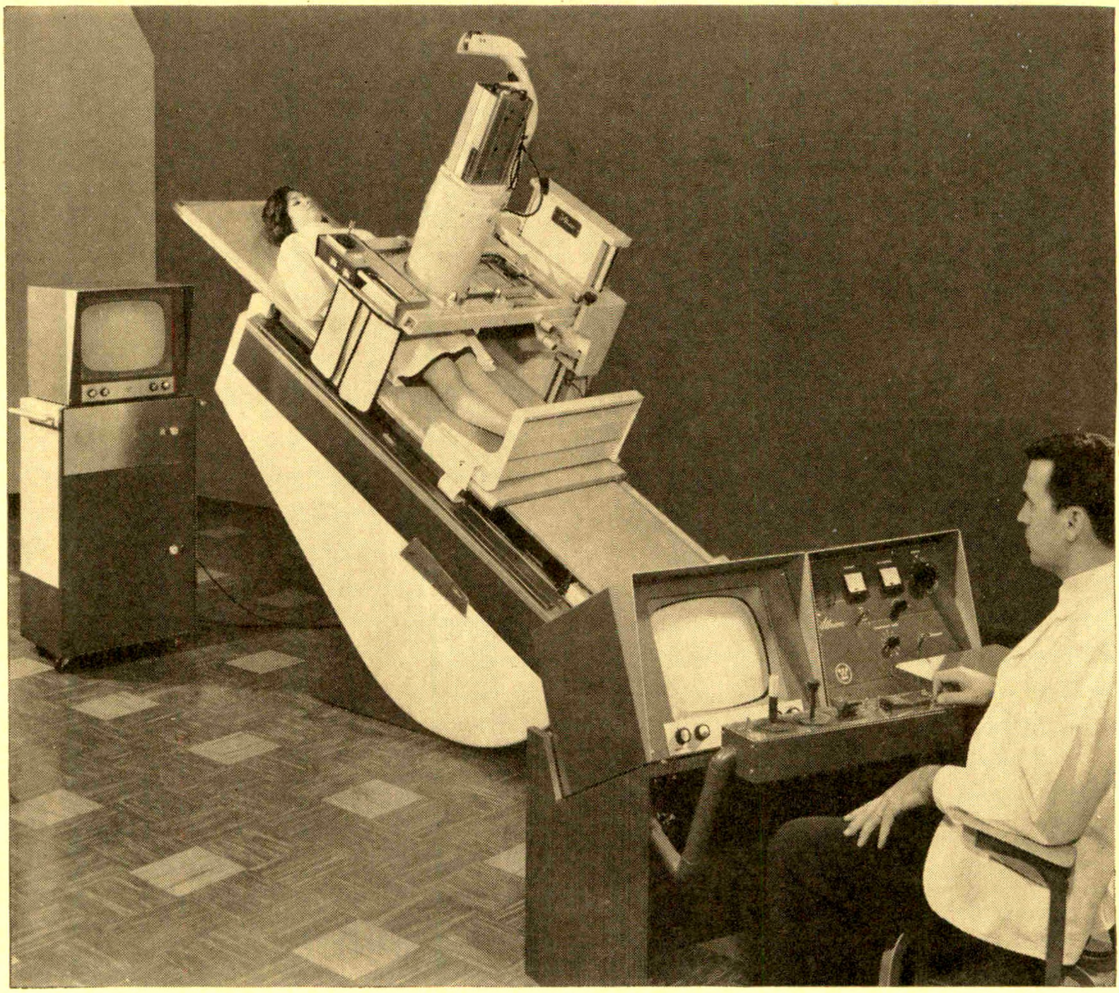

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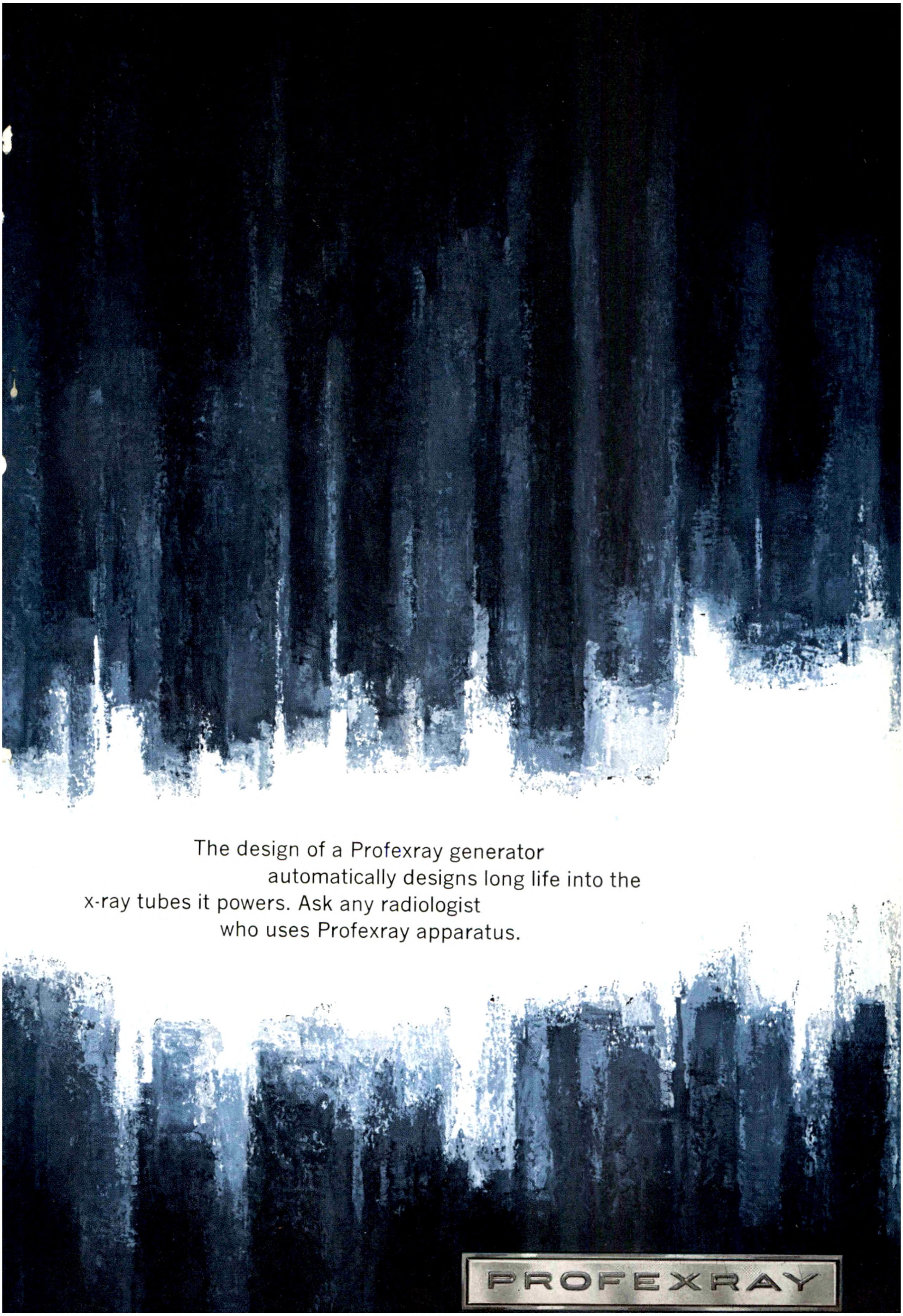

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Vol. 92 December, 1964 No. 6

CONTENTS

First a Radiologist; The President's Address. Harold O. Peterson, M.D. . . 1227

The Normal Internal Auditory Canal. Galdino E. Valvassori, M.D., and Robert H. Pierce, M.D. . . . 1232

A New Method of Pineal Localization. C. L. Oon, M.B., B.S., D.M.R.D. . 1242

Calcification in Intracranial Metastases. D. Gordon Potts, M.D., and Gerhart T. Svare, M.D. . . . 1249

The Interhemispheric Subdural or Falx Hematoma. Paul B. Wollschlaeger, M.D., and Gertraud Wollschlaeger, M.D. . . . 1252

The Incidence of Acquired and Congenital Fusions in the Cervical Spine. Michael W. Brown, M.D., Arch W. Templeton, M.D., and Fred J. Hodges, III, M.D. . . . 1255

Cervical Spondylotic Myelopathy Studied by Air Myelography. Paul H. Crandall, M.D., and William N. Hanafee, M.D. . . . 1260

Tumors of the Spine and Their Relationship to the Intervertebral Disk; A Roentgenographic-Anatomic Correlation. Alex Norman, M.D., and Catherine P. Kambolis . . . 1270

The Place of Diskography in Radiology As Based on 2,320 Cases. S. B. Feinberg, M.D. . . . 1275

Intraosseous Vertebral Venography in the Diagnosis of Lumbar Disk Disease. Louis A. Finney, M.D., Fredie P. Gargano, M.D., and August Buermann, M.D. . . . 1282

Spinal Lipoma. Fouad A. Halaby, Maj. MC, USA, Richard B. Peterson, Capt., MC, USA, and Robert C. Leaver, Maj. MC, USA . 1293

Multiple Lesions of the Spinal Cord. G. Lombardi, M.D., and A. Passerini, M.D. . . . 1298

Bone Island. Seuk Ky Kim, M.D., and William F. Barry, Jr., M.D. . 1301

The Fractured Ulnar Styloid; Roentgen Diagnosis of Recent Isolated Sprain Fracture. Edmund W. Klinefelter, M.D. . . . 1307

Camptodactyly. Guido Currarino, M.D., and Irvin Waldman, M.D. . 1312

(*Continued on page iv*)

Arthrography and Arteriography in Pigmented Villonodular Synovitis of the Knee. BURTON I. REIN, M.D., LARRY P. BILODEAU, M.D., AND PAUL JOHANSON, M.D. 1322
Poststenotic Dilatation (Aneurysm) of the Subclavian Artery Associated with Cervical Rib; Report of Two Cases. ISRAEL STEINBERG, M.D. . . . 1328
Posterior Cardiac Prominence in Ebstein's Anomaly Simulating Mitral Disease. DANIEL J. HANSON, M.D., AND HAROLD D. ROSENBAUM, M.D. 1331
Ebstein's Anomaly of the Tricuspid Valve; Angiocardiographic Considerations. KENT ELLIS, M.D., SYLVIA P. GRIFFITHS, M.D., JOHN O. BURRIS, M.D., G. CRAIG RAMSAY, M.D., AND RICHARD J. FLEMING, M.D. 1338
Arteriosclerotic Aneurysms of the Thoracic Aorta; Intravenous and Selective Visualization. ISRAEL STEINBERG, M.D., AND MORDECAI HALPERN, M.D. 1353
Ornithosis; Report of a Case. PAUL M. KROENING, MAJOR, MC 1370
Simulated Infiltration of the Lung Due to Pleural Thickening. SEWELL S. GORDON, M.D., AND MILTON GILCHRIST, M.D. 1373
Some Normal Renal Variants. JOHN L. DOPPMAN, M.D., AND ROBERT SHAPIRO, M.D. 1380
Infusion Pyelography. JOHN H. HARRIS, M.D., AND JOHN H. HARRIS, JR., M.D., D.Sc. (MED.) 1391
The Value of the Urea Washout Test in the Assessment of Curable Renovascular Hypertension. ROBERT E. STEJSKAL, M.D., EDWARD V. STAUB, M.D., MERLE K. LOKEN, PH.D., M.D., AND KURT AMPLATZ, M.D. . 1397
Officers 1409

Editorial

The Sixty-Fifth Annual Meeting of the American Roentgen Ray Society. K. L. KRABBENHOFT, M.D. 1410
Scientific Exhibits 1413
Technical Exhibits 1423
News Items 1425
Book Review 1428
Books Received 1428
Abstracts of Radiological Literature 1430
Subject Index to Volume 92 1443
Author Index to Volume 92 1457

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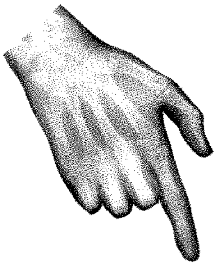

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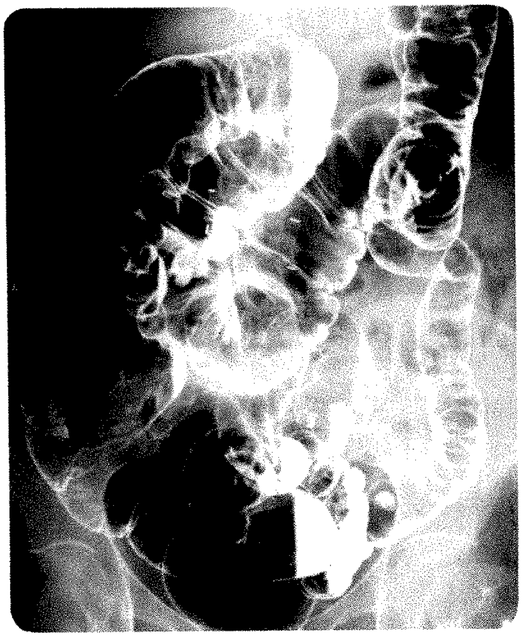

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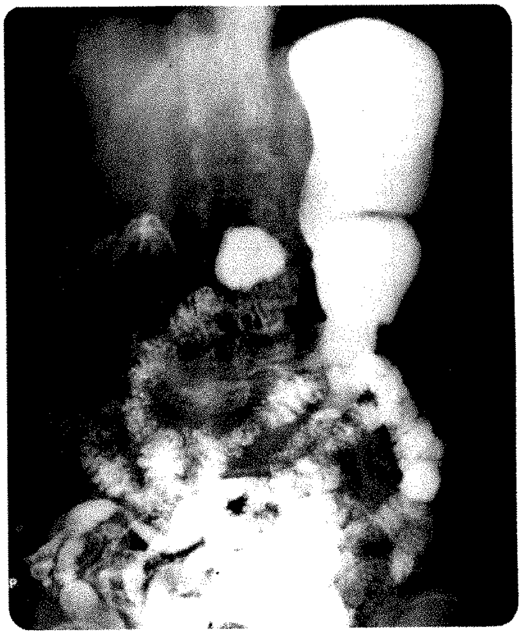

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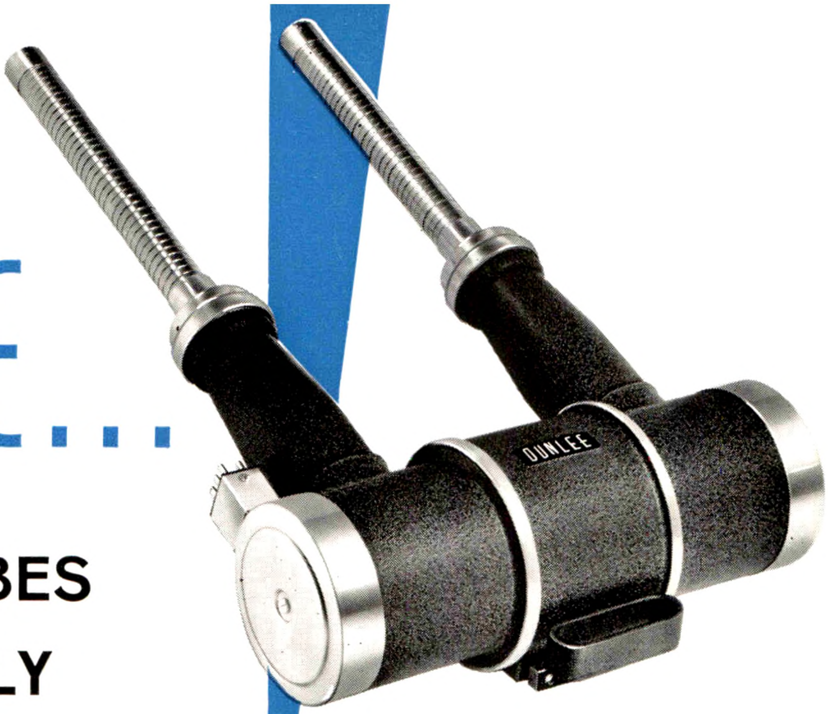

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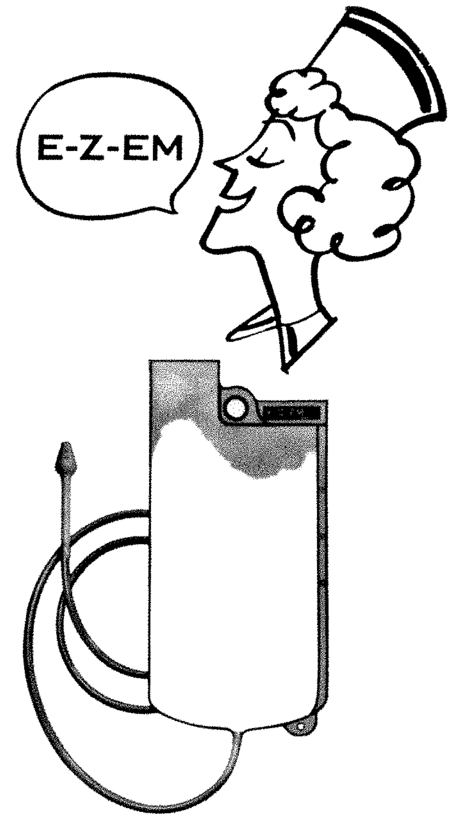

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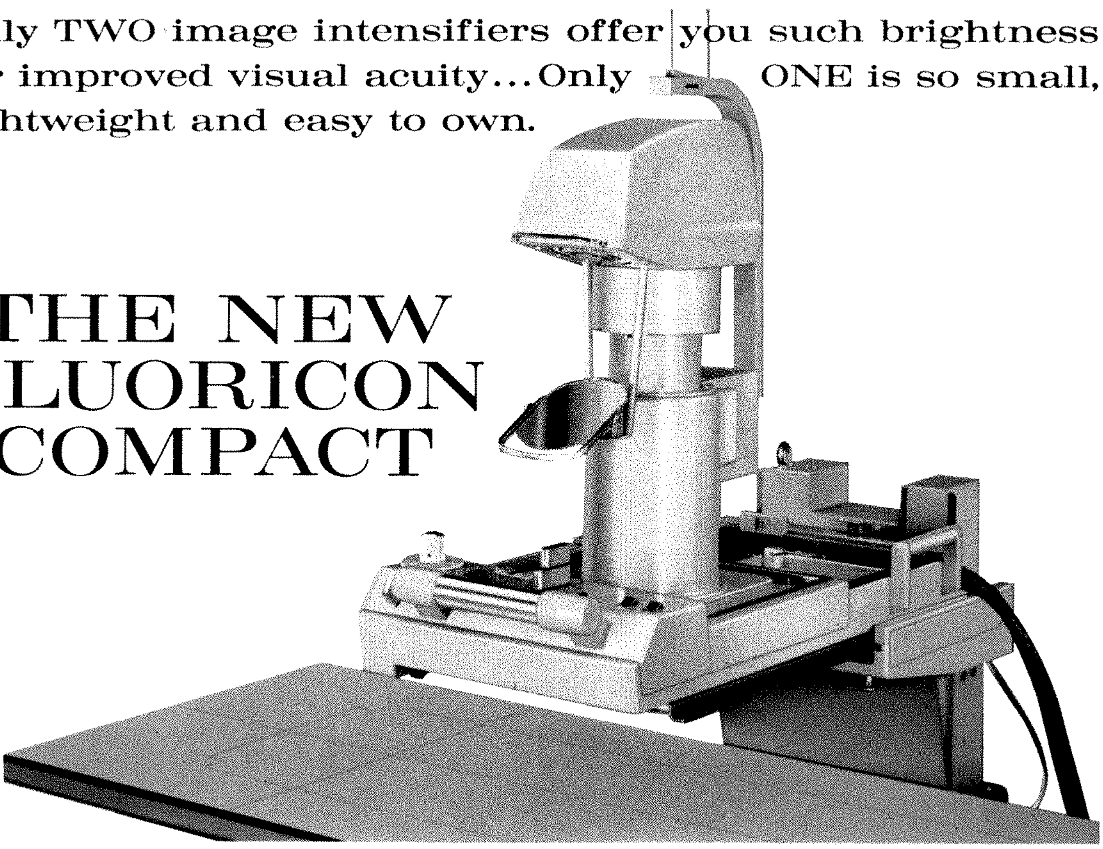

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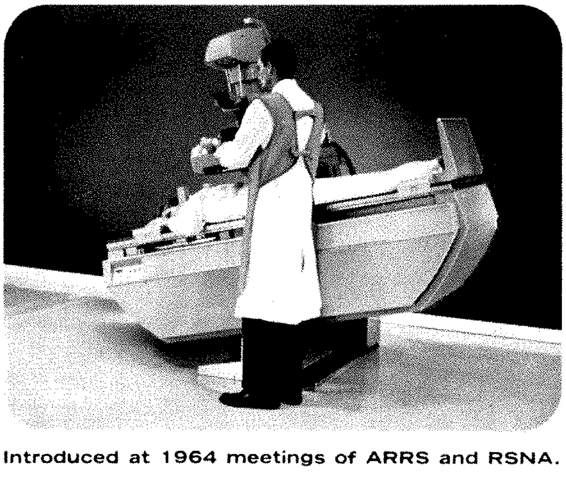

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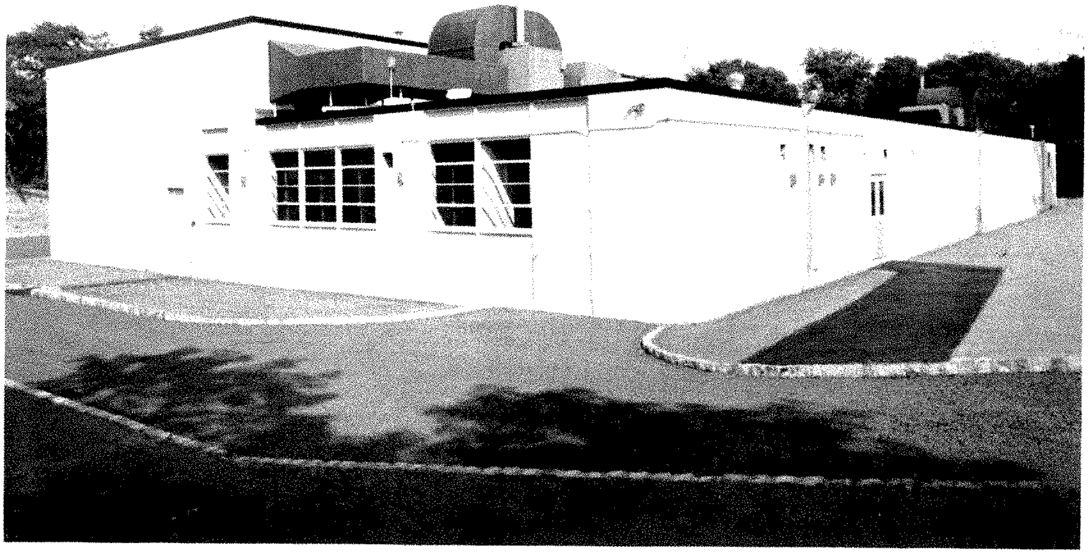

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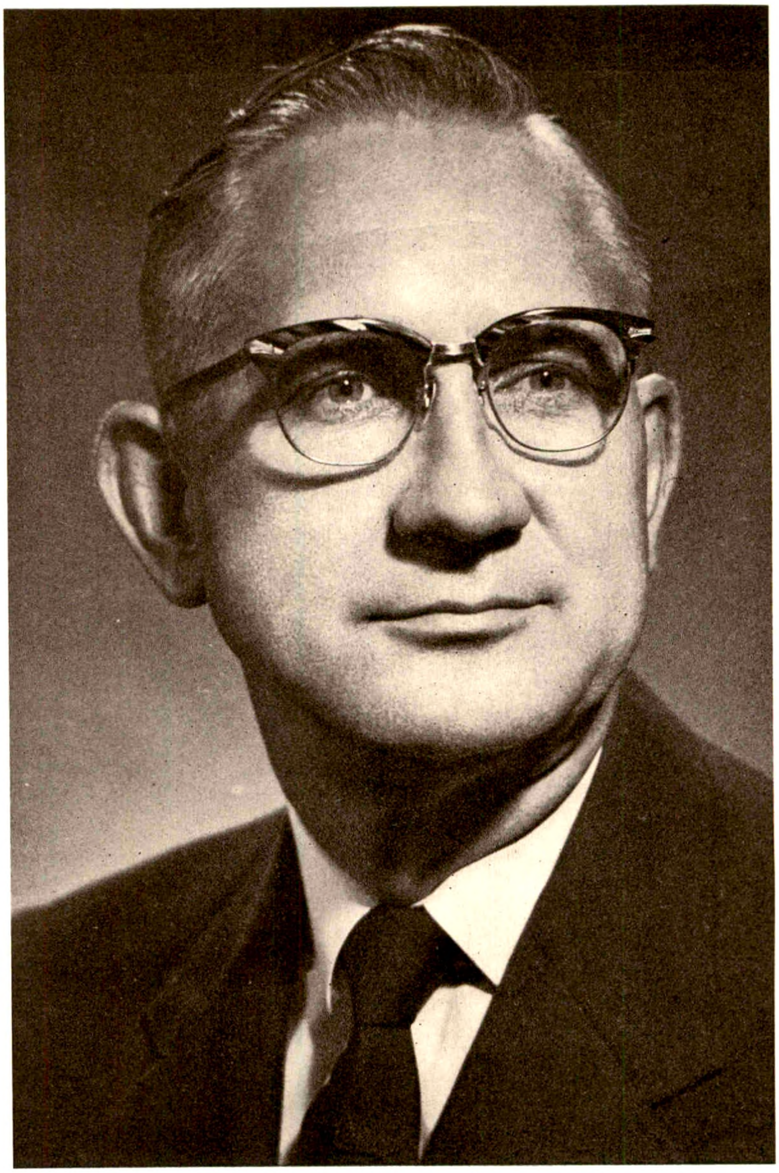

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VOL. 92 DECEMBER, 1964 No. 6

FIRST A RADIOLOGIST*

THE PRESIDENT'S ADDRESS

By HAROLD O. PETERSON, M.D.
MINNEAPOLIS, MINNESOTA

THE preparation of this Address has proved to be a most difficult task. To decide, even in a general way, what the topic should be has preoccupied my thinking for almost a year. After a greal deal of consideration I finally chose the title "First a Radiologist." This may sound like a take-off on the motion picture "First a Physician" with which I am sure most of you are familiar. This is, in a sense, correct although the motion picture was not the basis for selecting the subject matter.

I have selected this subject for two reasons. *First* and perhaps most compelling: I could think of no other subject. *Second:* I have long been of the opinion that radiologic diagnosis should be based solely on radiologic findings and that clinical findings, including the history, physical and laboratory studies, should play almost no role in the *final* radiologic diagnosis.

I have had many misgivings about trying to present this subject and have often considered abandoning it for something entirely different. The vacuum which always developed finally convinced me I had no choice. At the risk of being misunderstood and misquoted and losing most, if not all, of my friends in Radiology, I finally decided to proceed with the theme of "First a Radiologist." If I were naive enough to think that my opinions carried any weight, I might be setting Radiology back many years, but I know that this cannot be a valid criticism.

My remarks will pertain only to diagnostic radiology and have no relevance to therapeutic radiology nor to the newer diagnostic techniques utilizing other modalities such as isotopes, ultrasonics, thermography and palencephalography.

Whereas chronologically it is true that we are first trained in the basic principles of medicine the same as are all other Doctors of Medicine, following internship the training and experience of different physicians vary tremendously. By the time we, who have chosen Radiology, have passed the examination of the American Board of Radiology, we have spent at least as much time in Radiology as in our entire course in Medical School. We are now, by virtue of this special training, well on the road to becoming experts in the science of Radiology. Hence, by the title "First a Radiologist," it is meant to emphasize the fact that we are in truth radiologists and as such should first completely exhaust our own diagnostic methods extracting every bit of information possible from each procedure we do without utilizing other kinds of information to arrive at *our* final diagnosis.

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* Presented at the Sixty-fifth Annual Meeting of the American Roentgen Ray Society, Minneapolis, Minnesota, September 29–October 2, 1964.

Unfortunately, an unrealistic concept of a radiologist has developed, chiefly among radiologists, which portrays him as a *good* general all-around clinician with considerable knowledge concerning all of the specialties of medicine including all clinical laboratory procedures together with a comprehensive knowledge of and classification of all diseases and often times a good recommendation on the best treatment. As a stimulus towards perpetuating this myth, we are constantly exhorted by our peers and teachers in Radiology to examine the patient, take our own history and correlate all of these with the roentgen examination. I recall vividly Dr. Sossman's advice to a group like this to re-acquaint ourselves with an ancient and honorable medical instrument, the stethoscope, and use it every time we examine a heart. For me, and I venture to say for many of you, this approach to cardiac roentgenology could only be time consuming and misleading. Furthermore, I have found that clinicians who refer cases to us usually do not appreciate our relating to them our concept of the patient's history or what we found on physical examination. They generally desire, and quite properly expect, a good accurate radiologic diagnosis based on radiologic findings.

As a result of this all encompassing approach to Radiology it seems that often times when we consult with a clinician about his patient we first quiz him about the history and physical findings instead of intently studying the film. If a casual glance at the film plus "the revered correlation" with the clinical findings does not produce a ready diagnosis, we astutely inquire about the blood porcelain level. It almost seems that this intense interest in the clinical and laboratory picture serves primarily as a stall and a cover-up for inadequate Radiology. Furthermore, in using these tactics it becomes much too easy to grasp and cling to some lead in the clinical picture and project this into the film interpretation. Later, if the diagnosis proves to be incorrect, we can justify our error by placing at least part of the blame on the perplexing clinical picture. The net result is poorer radiology and a poorer image of radiologists among those who refer patients to us. It reminds one a bit of the old excuse "I read the films wet." Without having made any attempt at controlled testing, I have never felt that there was any evidence which proved that a wet film reading should automatically be a poor one other than the fact that there is a tendency in this situation to do a hurried and inadequate study of the film.

Another manifestation of the "clinical approach" in diagnostic radiology is to complain bitterly about the x-ray examination request which contains no clinical information. I am convinced, however, that the films usually could be better analyzed without a history provided they were carefully examined. There would surely be less over-reading, and, most likely, less significant findings overlooked. As one of my teachers in Radiology often said, "everything is on the film." We simply have to perceive and then analyze correctly all the shadows.

Should one then completely ignore the entire clinical picture? Certainly not. The more one knows about the patient and about all the various specialties in medicine including laboratory procedures, the better radiologist one can be, provided he realizes his severe limitations in clinical medicine and tries to get the maximum information out of his radiologic examination. The clinical picture may well bring to mind valuable possibilities which should result in even a more careful scrutiny of certain areas, but, in spite of this, when the final diagnosis is made it should be based on radiologic findings. To completely eliminate from consideration all clinical evidence would take much of the interest and sparkle out of diagnostic radiology and I do not recommend it. The final analysis of the films and the total extent of the roentgen study should be guided by all available pertinent clinical information. Nevertheless, the resultant radiologic diagnosis, if it is to be accurate, unbiased, and represent the independent judgment of the radiologist, must be based on radiologic findings. *The more the clinical findings are required in making a "radiologic" diagnois, the less valuable is the radiologic aspect of the diagnosis.* This is a truism even when the clinical findings are accurate. It is doubly true when there is a conflict of opinions concerning the clinical picture, which I believe is not an uncommon occurrence especially in teaching centers. *If the x-ray diagnosis can be changed from one conclusion to another because of the clinical picture, it is very often an unsatisfactory diagnosis.*

The x-ray diagnosis *per se* is undoubtedly the most accurate and this approach should be developed to the utmost. I have long remembered Hampton's oft repeated question at film conferences "Why don't you examine the patient?" What he meant was that the x-ray examination was inadequate for one reason or another and the case had not been properly and completely studied radiologically. When the radiologic examination is thorough and meticulous, it is often possible to make a really positive report such as Sossman once described in our Continuation Center Course, "The gallbladder is functioning and contains exactly four gallstones, indubitably, by God."

The radiologist should try to interpret each case with a deep conviction that when he makes a final diagnosis it must be correct. He cannot permit a wrong diagnosis. In this respect, his diagnoses should be comparable to those of the anatomic pathologist as closely as the radiologic method permits. This aim to be accurate and correct, while important in all branches of radiology, is particularly essential when the roentgen diagnosis may play a major role in deciding the treatment; *e.g.*, the diagnosis and localization of a brain tumor or a less complicated situation such as the diagnosis and localization of a symptomatic herniated disk.

On the other hand if it is impossible to make a diagnosis this, too, should be clearly stated. There are times when it may be necessary to follow the usual procedure of the clinician and simply list multiple possibilities hopefully in the order of likely probability. There are also times when one must clearly straddle the fence; this can be a very acceptable radiologic posture. There is no virtue in the old saying "I may be wrong but I am never in doubt." This is a totally unacceptable attitude even though we sometimes are complimented for taking a stand—right or wrong.

Before taking any stand we should always ask ourselves "What will this diagnosis mean to the patient—what will happen to him because of this opinion?" Although it is true that the ultimate responsibility for the care of the patient always rests, as it should, with the clinician in charge, we cannot abrogate our responsibility to the physician and the patient for any part of the radiologic examination. The clinician will regard our diagnoses as extremely important only if we continually strive to leave no room for error.

The fact that some clinicians in the so-called minor specialties have relatively little regard for our radiologic assistance in their field is due largely to the fact that we do not consistently produce accurate roentgen diagnoses. The clinician under these circumstances comes to rely more and more on his own ability to interpret the roentgen studies. Because his field of radiologic interest encompasses only a small branch of radiology, he surely should be able to do a superb job not because he knows the history and physical findings but primarily because he knows the anatomy and pathology of his particular field very well. However, he usually does not become a superb radiologist, partly because of the great difficulty he has in avoiding the influence of the clinical picture. He cannot render an impartial, independent evaluation of the roentgen examination alone. Conversely, the radiologist who should be able to make a straight forward analysis of the roentgen findings in terms of anatomy and pathology all too often fails, partly because he insists on disposing of his greatest asset, the pure radiologic approach, in order to admix the ingredients of the clinical picture. He also may fail because of incompetence in the radiologic specialty and no amount of self adulation will overcome this deficit.

Unfortunately, since most of us are a type of general practitioner, we are not likely to be expert in all branches of radiology. This should make it all the more apparent that we cannot be clinicians in all the various specialties in addition to being good radiologists in every specialty. The answer in large medical centers is, of course, to have specialists within the specialty of radiology and, indeed, this has been done for some time, particularly in such fields as neuroradiology, pediatric radiology and cardiovascular radiology. We are in need of many more sub-specialists in radiology, but, at the same time, we need to train an increasing number of generalists who have a more adequate knowledge of the specialties. Furthermore, this training must continue throughout our years of radiologic practice. Since there is so little time and so much to learn, it would assuredly help if we would concentrate on anatomy, pathology and some physiology as depicted by radiologic methods, thereby strictly observing the facts and eliminating as much as possible our inadequate attempts at clinical diagnosis.

Occasionally, in certain limited areas, largely because of some unusual volume of experience or very special interest, a radiologist might become a partly accomplished clinician, but by no stretch of the imagination does the usual training or experience of the radiologist make him a good clinician in all fields of medicine. As a consequence of this limited clinical ability, radiologic studies for the most part should not be ordered by the radiologist. This is particularly true in the area of special procedures. For example, the question whether a certain patient should have a myelogram or an angiocardiogram can properly be discussed or advised by the radiologist, but the physician who has *ultimate* responsibility for the patient should make the final decision. Once it has been determined that a special procedure is indicated, the best radiologic approach for accomplishing this purpose might properly be the function of the radiologist.

I regard myself to be a clinical radiologist. I have a real interest in the patients as individuals and try to keep this thought in mind as each diagnosis is made. Nevertheless, I believe that better radiology can be achieved by emphasizing the importance of a superior type of pure radiologic study and if necessary de-emphasizing the clinical findings. This will, of course, include the recognition of nonsignificant findings, thus cutting down on over-reading and subsequent needless additional procedures.

Do I like to know the clinical picture? I certainly do. Radiology would be terribly dull if we did not think in terms of the individual patient and his signs and symptoms, treatment, prognosis and results. However, I strive to eliminate the bias of this information from the roentgen report and am not dismayed when no clinical information is available. It simply means that a better analysis of the films will be necessary which, in turn, often means a better diagnosis.

The image of the radiologist will not be enhanced if we embark on lengthy discussions of clinical findings and conclude with wrong diagnoses. I doubt that we can elevate our image in anyone's opinion by simply proclaiming our status as physicians. Our image in the eyes of our confreres in other branches of medicine and their patients will be good and respected only if we render consistently sound and accurate roentgen diagnoses.

It should be abundantly clear that the vast amount of medical knowledge now available cannot be encompassed by any one individual. It is likewise not possible for a radiologist to know everything even in his own field of diagnostic radiology. Specialization within the specialty of radiology will help to improve the quality of radiology, but such a goal is impossible for the vast majority of radiologists in this country. Perhaps the next best solution is to utilize to the fullest, as well as to improve, our own radiologic methods. Hence the title—"*First a Radiologist.*"

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THE NORMAL INTERNAL AUDITORY CANAL*

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> . . . "I venture to predict that within the next few years we will be able to diagnose these tumors while they are still within the internal auditory canal and perhaps an ideal operation may be developed to remove them at this early stage."
>
> Fowler, 1936[8]

THE new surgical approaches to the internal auditory canal which allow an exploration of its full length have opened to the surgeon the possibility of removing small tumors which may be completely limited to the internal auditory canal.[12] These approaches have the added advantage of enabling the surgeon to remove small tumors, while sparing the facial nerve (VII) and possibly the cochlear portion of the auditory nerve (VIII). For this reason, it has become increasingly important for the radiologist to have a thorough knowledge of the normal roentgenographic anatomy of the internal auditory canal in order to recognize those minimal changes which are indicative of a pathologic condition.

The purpose of this study has been: (1) to find the best technique available at this time for the examination of the internal auditory canal; (2) to evaluate the various characteristics of the internal auditory canal and establish the ones that are useful for diagnosis; (3) to establish the range of the variation of normal between the two sides of the same subject; and (4) to compare the values obtained in this study on living subjects with the values gathered by others who have studied the internal auditory canals of disarticulated temporal bones or dry skulls.

ANATOMY

The terms canal, meatus and porus are frequently used without proper selection. Porus means merely the opening of the canal. The term meatus is often employed to designate either the canal or the opening of the canal. In order to avoid confusion, the term meatus will not be used in this report.

The petrous pyramids lie in the base of the skull at an angle approximately 45° to the sagittal plane. The internal auditory canal enters the petrous pyramid from its posteromedial surface at the junction of the anterior two-fifths with the posterior three-fifths. The long axis forms a right angle with the sagittal plane of the skull and an angle of about 45° with the long axis of the petrous pyramids (Fig. 1). The porus, on the other hand, has a shape much like the bevel of a needle with its maximum diameter in the same axis as the petrous pyramid where it opens on the posteromedial aspect. The posterior, superior and inferior lips of the porus are prominent and made up of dense bone, while the anterior lip is usually poorly demarcated so that the anterior wall of the canal blends in smoothly with the posteromedial wall of the petrous apex.

The internal auditory canal in cross section usually has an oval shape with its height (vertical diameter) being slightly larger than its width (horizontal diameter). However, the shape of the canal may

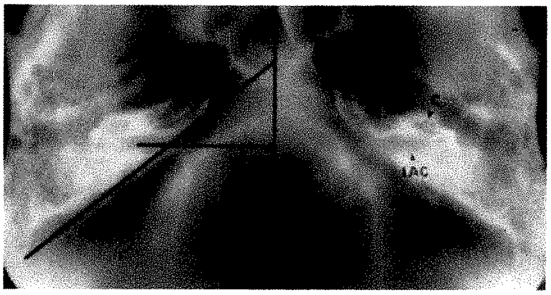

FIG. 1. Laminagram of a dry skull in axial projection showing relationship of the internal auditory canals and internal auditory pori to the petrous pyramids and the sagittal plane of the skull. C = cochlea; IAC = internal auditory canal.

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* From the Department of Radiology, The University of Chicago, Chicago, Illinois.

change at the porus due to the presence of variable lips from the longer anterior wall.

The internal auditory canal contains the facial nerve, the nervus intermedius, the acoustic nerve (which divides within the canal into its cochlear and vestibular portions), and the internal auditory branch of the basilar artery. The three nerves are all enclosed within a common sheath.

The lateral end of the canal is closed by a vertical plate (the *lamina cribrosa*). Arising from the *lamina cribrosa* is a horizontal crest (the *crista falciformis*) which divides the lateral part of the internal auditory canal into two unequal portions with the larger portion being the inferior one. Beneath the *crista falciformis* are three sets of foramina: the anterior set for the cochlear nerve, and the two posterior sets for the two branches of the inferior vestibular nerve, which terminate in the saccule and posterior semicircular canal. Above the *crista falciformis* are two sets of foramina: the anterior set for the facial nerve and nervus intermedius, and the posterior set for the superior vestibular nerve which terminates in the utricle, saccule and lateral and superior semicircular canals.

ROENTGENOGRAPHIC PROJECTIONS

From the above anatomic description of the internal auditory canal, one can deduce that the best projections for the study of the internal auditory canal are: the frontal and axial views for a demonstration of the canal in its full length, and the lateral for a visualization of the canal on end. Stenvers' views are unsatisfactory for the visualization of the internal auditory canal, which is markedly foreshortened due to its obliquity to the plane of the film. These views are useful for the demonstration of the porus, which is seen *en face*. Unfortunately, the study of the internal auditory canal in the above described projections by conventional roentgenography is far from satisfactory because of the superimposition of structures located outside as well as within the petrous pyramid, which may obscure the view of the canal. For example, the presence of large air cells in a very pneumatized petrous pyramid may obliterate the outline of the canal.

Laminagraphy, when performed with an apparatus furnishing a sufficiently thin cut and a good coefficient of distinctness, permits the use of the best anatomic projections and avoids the superimposition of undesired structures.

FRONTAL PROJECTIONS

By conventional roentgenography, the most satisfactory view of the petrous pyramids is an anteroposterior projection with the tube placed at such an angle that the internal auditory canals cast a well-defined band of radiolucency through the orbits. The canals extend from the medial wall of the vestibule (which is usually well seen as a radiolucent, round area from which the semicircular canals arise) medially to approximately 1.5 cm. from the petrous apex. Often the pneumatization in the petrous apex produces the false impression that the canal extends to the apex. The cochlea is partially superimposed upon the proximal portion of the canal, accounting for the less radiolucent appearance of this tract in comparison to the medial end.

The laminagrams demonstrate the internal auditory canal (Fig. 6) with its longer anterior wall and the shorter posterior wall whose medial, concave lip is sharply outlined (Fig. 2).

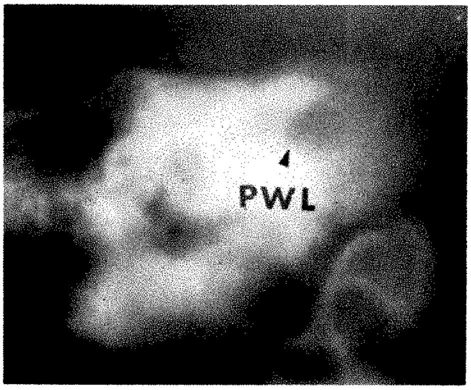

FIG. 2. Frontal laminagram showing the concave lip of the posterior wall of the internal auditory canal. PWL = posterior wall lip.

In the Towne's view, the 30° angulation of the roentgen-ray beam caudally modifies the projection of the petrous pyramids upon the roentgenogram in such a way that the upper portion of the posteromedial aspect of the pyramid forms the superior contour of it. In this way, the internal auditory canal is seen extending to the superior outline of the petrous pyramid, forming a dehiscence with its porus.

AXIAL PROJECTION

While on conventional submentum-vertex projection roentgenograms the internal auditory canal is poorly and incompletely visualized, the laminagrams clearly show the canal in its full length, as well as the anteroposterior width of the porus (Fig. 1).

LATERAL PROJECTION

The internal auditory canal is not seen in regular lateral view because of superimposed structures. With laminagraphy, the internal auditory canal is visualized in cross section (Fig. 3). At its medial end, the posterior wall disappears because of its shorter length when compared with the anterior wall. The superior and inferior lips of the porus are well demonstrated (Fig. 4).

STENVERS' PROJECTION

In the regular Stenvers' view, the porus and canal form an oval-shaped radiolucency which opens medially. The medial portion is formed by the porus and the lateral portion by the canal, which is markedly foreshortened due to its obliquity to the plane of the film. The superior and inferior lips of the porus are clearly outlined, while the anterior and posterior lips are not well seen. This gives one the impression that only the canal is visualized. The porus lies about 2.0 cm. from the tip of the petrous pyramid. The pneumatization in the petrous tip should not be confused with the medial end of the canal.

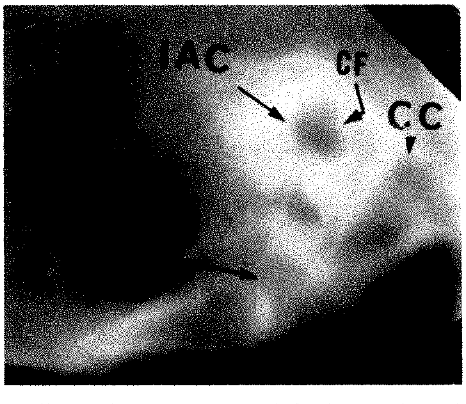

FIG. 3. Lateral laminagram of the internal auditory canal in its lateral portion. Note the crista falciformis bisecting the canal. IAC = internal auditory canal; CC = carotid canal; JF = jugular fossa; CF = crista falciformis.

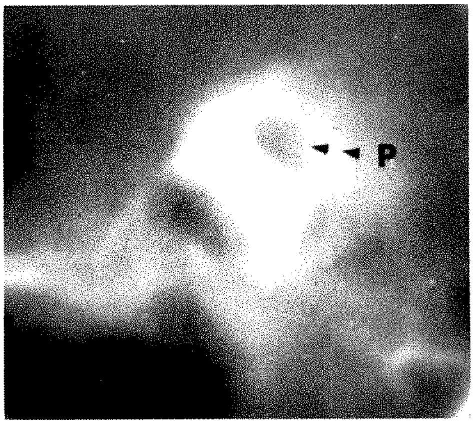

FIG. 4. Lateral laminagram of the internal auditory porus showing the "C" configuration which is present after termination of the posterior wall. P = porus.

Laminagrams in Stenvers' projection clearly demonstrate the anatomy. The internal auditory canal is well seen as an oval foramen in the posterior sections of the petrous pyramids. The porus, which in this projection is located posteromedial to the canal, appears as a dense "C" open medially, where the anterior lip is less prominent (Fig. 5).

REVIEW OF THE LITERATURE

In order to establish the roentgenographic appearance, measurements and variation of "normal," several authors have studied the internal auditory canals in the dry skull and in histologic sections.[2,6,10]

Ebenius,[6] in 1934, reported on a series of 100 dry skulls in the Towne and axial projections, which he considers the most suit-

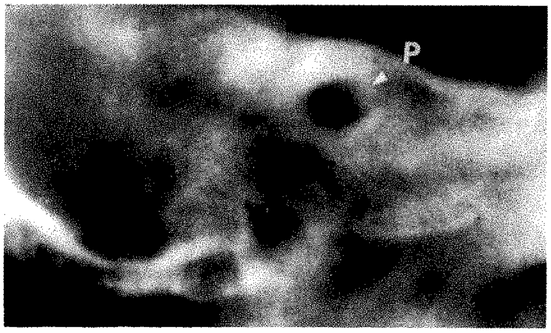

Fig. 5. Laminagram in Stenvers' projection of the internal auditory porus. P = porus.

able for this study. He found the vertical diameter of the canal to range from 5 to 9 mm. with an average of 6.3 mm. He found the horizontal diameter (or width) to range from 4 to 8 mm. with an average of 5.7 mm. These measurements have a 28 per cent magnification in the frontodorsal projection and a 25 per cent magnification in the axial projection. By comparing the internal auditory canals of the two sides of 100 dry skulls, he found 96 per cent with a variation of up to 1.0 mm., 3 per cent with a variation from 1.0 to 2.0 mm., and 1 per cent with a variation between 2.0 and 3.0 mm.

Camp and Cilley,[2] in 1939, reported on the study of 509 disarticulated petrous bones, 250 of which were paired. These authors found the anteroposterior projection to be the most desirable. They divided the canals into four different shapes: straight (when no more than a 0.5 mm. variation existed throughout the canal's length) in 60 per cent of the cases; narrowed medially (when the lateral end was more than 0.5 mm. larger than the medial end) in 12 per cent of the cases; narrowed laterally (when the medial end was more than 0.5 mm. larger than the lateral end) in 16 per cent of the cases; and oval (when the mid-portion was greater than either end of the canal) in 12 per cent of the cases. In comparing the 250 petrous bones of the two sides, the shape of the canal was the same in 70 per cent. The vertical diameter of the canal varied from 2.5 mm. to 11 mm. with the average of 5.23 mm. However, only two internal auditory canals measured more than 9.0 mm. Comparison of the internal auditory canals of the 250 paired bones showed 75 per cent with no more than a 1.0 mm. variation, 23 per cent with a variation from 1.0 to 2.0 mm., and 2 per cent with a variation from 2.0 to 2.5 mm. The length of the posterior wall of the canal was measured in 100 pairs of petrous bones with the length varying from 3.0 to 16 mm. and having an average of 7.8 mm.; 27 per cent had a variation of up to 1.0 mm., 38 per cent had a variation from 1.0 to 2.0 mm., 7 per cent had a variation from 3.0 to 4.0 mm., and only 1 per cent had a variation from 4.0 to 5.0 mm. According to Camp and Cilley,[2] all of the measurements in their study were taken from roentgenograms having negligible magnification due to their technique.

Graf,[10] in his monograph published in 1952, studied the internal auditory canals by histologic sections in dry bones and by roentgenograms of living subjects. He also studied the internal auditory porus in the dry bones. In 60 horizontal histologic sections, he found the horizontal diameter to range from 3.0 to 5.0 mm., with an average of 4.0 mm.; in 46 vertical histologic sections, he found the diameter to range from 3.0 to 6.5 mm., with the average being 4.7 mm.; and in 56 horizontal sections he found the length of the internal auditory canal to range from 5.0 to 11 mm., with the average being 8.0 mm. In 66 dry bones, he found the vertical diameter of the canal to vary between 3.5 and 8.0 mm., with the average of 5.0 mm.; and on these same bones the vertical diameter of the porus ranged from 2.5 to 6.0 mm., with the average of 5.0 mm. In comparing the porus with the canal, Graf found that the vertical diameter of the canal was most commonly 1.0 mm. larger than the porus. However, in some cases it was up to 3.0 mm. larger. *In no case was the porus larger than the canal.* In 100 living subjects, he also measured the vertical diameter of the canal in the Stenvers' projection and found a range from 3.0 to 7.0 mm., with an average of 4.8 mm.

METHOD AND MATERIAL

The University of Chicago study was performed on 300 temporal bones of living subjects, of which 200 were paired. One hundred and twenty-five of these bones were from 100 patients seen at The University of Chicago for otosclerosis, and the measurements were made from the preoperative examinations. The remaining 175 temporal bone measurements were obtained from 100 patients having roentgenologic examinations of the temporal bone for conditions not referable to the internal auditory canals.

The parameters which were investigated in this study included: shape, vertical diameter, length of the posterior wall, length of the anterior wall and position of the crista falciformis.

Because it has been claimed by some authors that otosclerosis can produce a narrowing of the internal auditory canals,[4] the measurements from patients with this disease were compared with those obtained from patients with no known otosclerosis (Table III).

All the measurements of this study: (1) have been obtained from anteroposterior roentgenograms with the patients positioned so that the line from the external canthus to the tragus was perpendicular to the plane of the film; (2) have been taken to the nearest millimeter; and (3) have been corrected for the 30 per cent magnification factor inherent in the Polytome.

Finally, the measurements were compared with those obtained by the authors mentioned above.

SHAPE

The shapes were classified with measurements taken just medial to the lamina cribrosa, at the termination of the posterior wall, and at the point of greatest variation, if any, between these two points (Table I).

There were three different shapes noted in this study: straight, narrowed medially and oval. A canal was considered straight when there was no more than 1.0 mm. difference in the diameter anywhere in its length (51 per cent of the cases) (Fig. 6); a canal was considered narrowed medially when the diameter of the medial end was at least 1.0 mm. smaller than any other portion of the canal (30 per cent of the cases) (Fig. 7); and a canal was considered oval when the diameter of its mid-portion exceeded by over 1.0 mm. both the medial and the lateral ends (19 per cent of the

TABLE I
SHAPE

		University of Chicago Series (per cent)	Camp and Cilley[2] Series (per cent)
Shape	Straight	51	60
	Narrowed Medially	30	16
	Narrowed Laterally	—	12
	Oval	19	12
Bilateral Comparison	Same	90	70
	Different	10	30

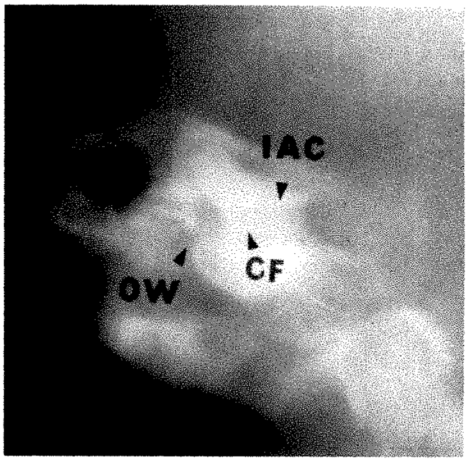

FIG. 6. Frontal laminagram showing a straight internal auditory canal. IAC = internal auditory canal; CF = crista falciformis; OW = oval window.

cases) (Fig. 8). Camp and Cilley[2] found, besides these three shapes, a fourth which was considered to be narrowed laterally. This latter classification accounted for 12 per cent of their series. None of the canals studied in The University of Chicago series had this shape. This discrepancy may be accounted for by the following factors: (1) 1 mm. was used in this series for determining shape differences, whereas Camp and Cilley[2] used 0.5 mm. In this way, canals considered straight by us may have been classified as narrowed laterally by them; and (2) laminagraphy was used in this series which allows a very distinct outline of the entire canal which is, instead, partially obscured in conventional roentgenograms.

When the 100 paired internal auditory canals were compared in our study, the shape was the same in 90 per cent of the cases. In the other 10 per cent the variation in shape was due to a difference in diameter of no more than 2.0 mm. Camp and Cilley[2] found the shape to be the same in 70 per cent of their cases. Again we feel that this difference is mostly due to our classification, which is based on 1.0 mm. variation rather than 0.5 mm. variation.

VERTICAL DIAMETER (HEIGHT)

This was measured in a plane perpendicular to the long axis of the canal (Table II). The maximum and minimum diameters were recorded. The vertical diameters of the internal auditory canals in our series varied from a maximum of 8.0 mm. to a minimum of 2.0 mm., with the average being 4.0 mm. The vertical diameter of the canal at the level of the lamina cribrosa is almost constant, ranging from 4.0 to 5.0 mm. When our measurements are compared with the values obtained by Ebenius,[6] Camp and Cilley,[2] and Graf,[10] there is a remarkable degree of consistency if one remembers that the factors used by Ebenius[6] need to be corrected for the 25 per cent magnification factor.

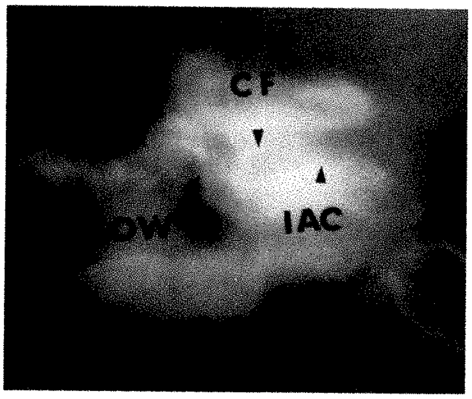

FIG. 7. Frontal laminagram showing an internal auditory canal which is narrowed medially. OW = oval window; CF = crista falciformis; IAC = internal auditory canal.

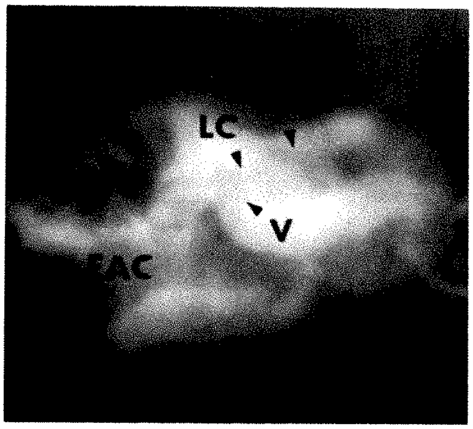

FIG. 8. Frontal laminagram showing an oval internal auditory canal. EAC = external auditory canal; LC = lamina cribrosa; V = vestibule; IAC = internal auditory canal.

A comparison between the internal auditory canals of the same subject in our series shows that 99 per cent of the patients had only 1.0 mm. or less difference between the two sides. The remaining 1 per cent had a variation of up to 2.0 mm.

In Table II it is seen that Camp and Cilley[2] have only a 75 per cent incidence of canals which vary by 1.0 mm. or less, 23 per cent incidence of canals which vary between 1.0 and 2.0 mm., and a 2 per cent incidence varying from 2.0 to 2.5 mm. Again, it is believed that this discrepancy is more apparent than real, because measurements were taken only to the nearest millimeter in The University of Chicago study.

It is interesting to note, in reviewing all the series mentioned above, that only three

Table II

VERTICAL DIAMETER (HEIGHT)

		University of Chicago Series	Ebenius[6] Series	Camp and Cilley[2] Series	Graf[10] Series	
					Histologic	Dry Skull
Diameter	Maximum	8.0 mm.	9.0 mm.	11.0 mm.	6.5 mm.	8.0 mm.
	Minimum	2.0 mm.	5.0 mm.	2.5 mm.	3.0 mm.	3.5 mm.
	Average	4.0 mm.	6.3 mm.	5.23 mm.	4.7 mm.	5.0 mm.
Bilateral Variation	Up to 1.0 mm.	99%	96%	75%	—	—
	1 to 2 mm.	1%	3%	23%	—	—
	2 to 3 mm.	—	1%	2%	—	—

canals, two in the Camp and Cilley[2] and one in the Ebenius[6] series, showed a variation between the two sides of 2.5 mm.

In comparing the vertical diameters in our study, no significant difference was found between the otosclerosis and nonotosclerosis series (Table III).

POSTERIOR WALL

The length of the posterior wall was measured from the lamina cribrosa to the deepest portion of the concave lip of the posterior wall, which should always appear very distinct and regular (Table IV) (Fig. 2).

Table III

COMPARISON OF VERTICAL DIAMETER IN OTOSCLEROTIC AND NONOTOSCLEROTIC SERIES

		Otosclerotic		Assorted	
		No.	Per Cent	No.	Per Cent
Shape	Oval	24	19.2	32	18.3
	Narrowed Medially	45	36.0	45	25.7
	Straight	56	44.8	98	56.0
Diameter		mm.		mm.	
	Maximum	7.0		8.0	
	Minimum	2.0		2.0	
	Average	4.0		4.0	

In this series, the greatest length was 11.0 mm., the shortest length was 4.0 mm., and the average length was 8.0 mm. From Table IV it is seen that the average lengths are almost identical in this series, Camp and Cilley's[2] series and Graf's[10] series. On the other hand, the discrepancy between the maximum length is presumably due to the difficulty in distinguishing the posterior wall from the anterior wall in conventional roentgenograms. In support of this hypothesis is the fact that the maximum length of the posterior wall measured on a conventional roentgenogram is 5.0 mm. longer than the maximum length found by histologic direct measurements and by laminagraphy.

In our series, comparison between the posterior wall of the right and left canals of the same patient showed a variation of up to 1.0 mm. in 86 per cent of the cases, a variation of from 1 to 2 mm. in 13 per cent of the cases, and a variation of 2 to 3 mm. in only 1 per cent of the cases. When comparing these figures with those of Camp and Cilley,[2] a discrepancy is noted (Table IV). This discrepancy may be explained on the same basis as with the measurement of the maximum length of the canal.

Table IV

POSTERIOR WALL

		University of Chicago Series	Camp and Cilley[2] Series	Graf[10] Series	
				Histologic	Dry Skull
Length	Maximum	11.0 mm.	16.0 mm.	11.0 mm.	—
	Minimum	4.0 mm.	3.0 mm.	5.0 mm.	—
	Average	8.0 mm.	7.8 mm.	8.0 mm.	—
Bilateral Variation	Up to 1.0 mm.	86%	27%	—	—
	1 to 2 mm.	13%	38%	—	—
	2 to 3 mm.	1%	23%	—	—
	3 to 4 mm.	—	7%	—	—
	4 to 5 mm.	—	5%	—	—

LENGTH OF THE ANTERIOR WALL

The length of the anterior wall was measured from the lamina cribrosa to the most medial point of its inferior lip (Table v). This measurement was sometimes arrived at with difficulty because of the gradual blending of the anterior wall of the porus with the posteromedial aspect of the petrous pyramid.

The greatest length was 19 mm., the shortest length was 6 mm., and the average length was 12 mm. A comparison between the internal auditory canals of the same patient shows a variation up to 1 mm. in 85 per cent of the cases, a variation from 1 to 2 mm. in 8 per cent of the cases, and a variation from 2 to 3 mm. in 7 per cent of the cases.

As far as our review of the literature has shown, no other authors have measured the length of the anterior wall of the internal auditory canal.

An interesting anatomic point concerning the anterior wall is the presence of variable osseous lips protruding at the level of the porus, often narrowing this area considerably (Fig. 9). The significance of this finding is still under investigation.

Table V

ANTERIOR WALL

Length	Maximum	27.0 mm.
	Minimum	9.0 mm.
	Average	18.0 mm.
Bilateral Variation	Up to 1 mm.	85%
	1–2 mm.	8%
	2–3 mm.	7%

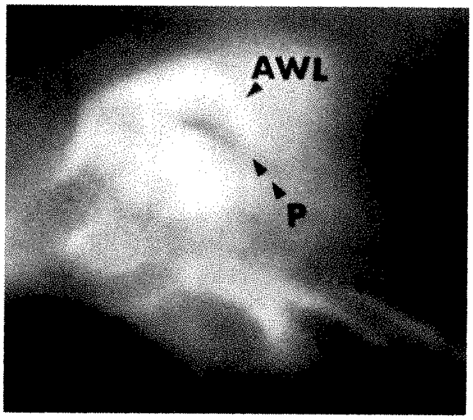

Fig. 9. Lateral laminagram showing a narrowing of the porus by a prominent lip extending from the anterior wall. AWL=anterior wall lip; P=porus.

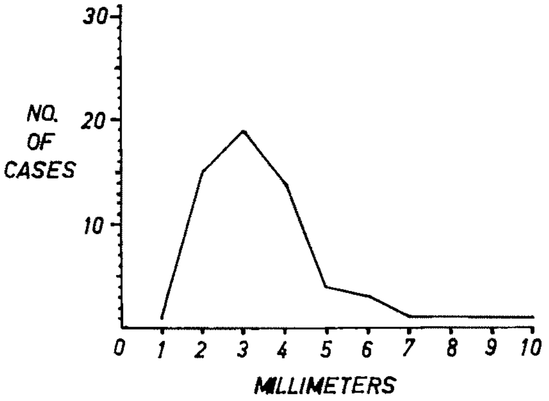

Fig. 10. A graph showing the length of the crista falciformis in millimeters from a random sampling of 100 petrous bones.

CRISTA FALCIFORMIS

One hundred petrous bones were surveyed for the study of the crista falciformis (Fig. 10). We have considered three factors: ratio of visualization, length, and position of the crista in relation to the vertical diameter of the canal.

The crista falciformis, or at least its origin, was visible in all petrous bones. In 85 per cent of the cases, the crista appeared as a linear density, ranging from 1 mm. to 7 mm. (Fig. 8). The thickness appeared to decrease progressively from its lateral origin to its medial end. In 15 per cent of the cases, only the origin of the crista was detectable as a definite knob of increased density.

The origin of the crista falciformis was located at, or above, the mid-point of the vertical diameter in all of the cases.

CONCLUSIONS

The above described study has led us to the following conclusions:

1. Laminagraphy should be considered the most satisfactory method for the study of the internal auditory canal. The frontal projection is the best projection for this purpose and offers the additional advantages of being extremely simple and allowing a simultaneous visualization of both internal auditory canals.

2. While the internal auditory canal of different subjects may vary considerably in shape, a variation in shape between the internal auditory canals of the same subject, which occurred in only 10 per cent of our cases, should suggest a further investigation.

3. A canal which appears narrowed laterally (whose medial end is 1 mm. or more larger than the remainder of the canal) should be considered abnormal.

4. Any canal with a vertical diameter under 2 mm. or over 9 mm. should be considered abnormal.

5. A variation of the vertical diameter between the two sides of the same patient of 1 mm. is considered insignificant, a variation from 1 to 2 mm. is considered questionable, and a variation of over 2 mm. is considered definitely abnormal. Of course, in comparing the internal auditory canals of the same patient it is extremely important to compare the same points of each canal.

6. The concave medial lip of the posterior wall should always be distinct and smooth (Fig. 2). The absence of this finding is of utmost significance.

7. The length of the posterior wall normally ranges from 4 to 11 mm.

8. A variation in the length of the posterior wall between the two sides by up to 2 mm. is considered insignificant, a variation from 2 to 3 mm. is considered questionable, and a variation of over 3 mm. is considered abnormal.

9. The length of the anterior wall is difficult to evaluate. It ranges approximately from 9 mm. to 27 mm.

10. A variation between the two sides in the length of the anterior wall of over 4 mm. should be considered abnormal.

11. The crista falciformis, or at least its origin, should be visualized in all cases (Fig. 8).

12. The length of the crista falciformis is extremely variable. However, its position in relation to the height of the canal at the lamina cribrosa is consistently at, or above, the mid-point. If the position of the crista falciformis lies below the mid-point, it should be considered abnormal.

It must be emphasized once again that all of the above measurements have been corrected for magnification and, therefore, represent the true anatomic size.

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* * *

A NEW METHOD OF PINEAL LOCALIZATION

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THE pineal body, which Descartes once thought to be the seat of the soul, is a small glandular structure, 8–10 mm. long, somewhat resembling a pine cone in shape. Recent biochemical researches have shown that the pineal body contains a group of active substances related to indole—the so-called "indole hormones." These include serotonin, melatonin and adrenoglomerulotropin.[10] The pineal body, therefore, deserves to be called the pineal gland.

Of roentgenologic importance is the fact that the pineal gland undergoes involution in childhood. During this process, calcification often occurs to an extent that the organ becomes visible on roentgen examination. The frequency of visualization of the pineal gland increases with advancing age and with improvement of roentgenographic techniques. Dyke,[3] in a survey of 3,000 consecutive skull roentgenograms, found the pineal body visible in about 60 per cent of adults.

The pineal gland occupies a central position in the skull and is, therefore, readily dislocated by a space-occupying intracranial lesion. The importance of a lateral pineal shift as a diagnostic aid, first pointed out by Schüller[12] in 1918, is now firmly established. However, the value of anteroposterior and vertical displacement in the localization of intracranial lesions is equivocal, largely through the lack of a satisfactory roentgenologic method of pineal gland orientation. Indeed, the numerous methods proposed for localizing the pineal gland on the lateral skull roentgenogram bespeak inherent shortcomings in each of them. And the method most popularly in use still remains the original technique proposed by Vastine and Kinney[16] in 1927.

AVAILABLE METHODS OF PINEAL LOCALIZATION

VASTINE-KINNEY METHOD

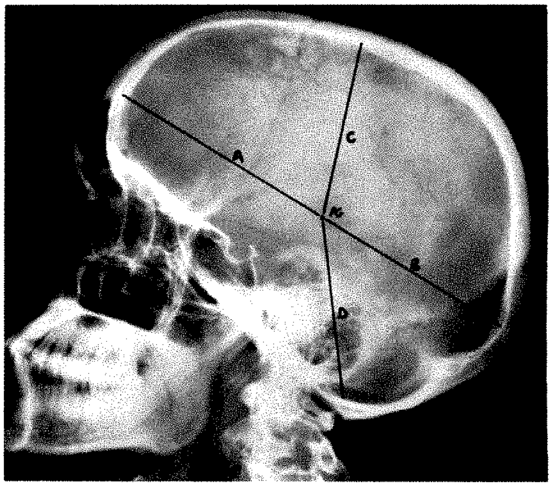

FIG. 1. Vastine-Kinney Method. A lateral skull roentgenogram showing the measurements used in this method. The distances are determined from the pineal gland to the inner table of the frontal bone (A) and to the inner table of the occipital bone (B) at their most distant points; to the inner table of the vault (C) and to the inner table of the cerebellar fossa near the foramen magnum (D). PG = pineal gland. These measurements are used in conjunction with charts such as shown in Figure 5, *A* and *B*.

Vastine and Kinney[16] described a method based on a graphic analysis of 200 normal cases (Fig. 1). They plotted the distance of the pineal gland from the frontal bone against the longitudinal or anteroposterior diameter of the skull, and the distance of the pineal from the vertex against the vertical or craniocaudal diameter of the skull. By thus determining the ranges for normal pineals, any position outside the normal zones was assumed to be the result of pathologic displacement.

DYKE'S MODIFICATION

Dyke[3] reviewed the Vastine-Kinney method and noted that a considerable number of normal pineal glands fell in front of the normal zone. To overcome this difficulty, he advised shifting this normal zone 4 mm. forward. The modification is unsatisfactory because it increases the number of normal pineal bodies falsely displaced posteriorly.

PROPORTIONAL METHOD OF FRAY

This method described by Fray[4] is an attempt to improve the Vastine-Kinney technique, and dispenses with the need to consult charts and make direct measurements with a rule. It utilizes an elastic band of uniform stretch with markers defining the normal zones. The elastic band is stretched successively along the anteroposterior and vertical diameters of the skull and normal pineals should fall within the segment equal to 55–60 per cent of the anteroposterior diameter measured from the frontal bone, and within the segment equal to 51–57 per cent of the vertical diameter measured from the vertex. In practice, however, this method fails to enhance appreciably the accuracy of pineal localization.

CRANIO-ANGLE METHOD

This procedure, also devised by Fray[5] avoids linear skull measurements and makes use of angles drawn on a transparent celluloid guide (Fig. 2). To determine displacement in the anteroposterior axis, the celluloid guide is so placed that the vertex of an 8° angle is at the posterior margin of the foramen magnum, the main base-line extending through the bregma. Pineal glands that are in normal position fall between the lines forming the angle. To ascertain displacement in a vertical direction, another angle of 11° is placed with its vertex at the base of the anterior clinoid processes and the main arm of the angle through the lambda. The normal pineal bodies fall within the angle. Fray himself stated that this method was not as accurate as others in determining anteroposterior displacement even though it might be useful in skulls of unusual shape.

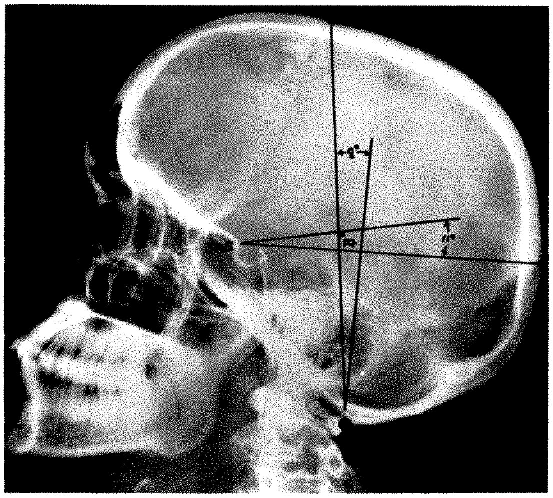

Fig. 2. Cranio-Angle Method. Two angles are utilized in this technique. To determine anteroposterior displacement of the pineal gland, an 8° angle is used with its base-line passing through the posterior margin of the foramen magnum and the bregma. For vertical pineal displacement, an 11° angle is employed with its base-line extending from the base of the anterior clinoids to the lambda.

LILJA METHOD

Lilja[7,8] in Stockholm made an interesting statistical study of pineal orientation in the skull by measuring distances between the pineal gland and numerous selected points in the cranium, including the Vastine-Kinney points. He expressed his data as ranges of normal variation in the ratios of skull-pineal distance (from the various end-points) to the anteroposterior and vertical diameters. This method has the distinct merit of indicating the statistical odds that a pineal body in a given location is normal or displaced. The objection to this method is that it is extremely time-consuming and hence impractical.

DE CRINIS AND RUSKEN METHOD

De Crinis and Rusken[1] in 1939 described yet another linear method of pineal localization. There are several serious objections to this method, which is not used to any great extent.

An excellent account of the various methods of pineal localization with critical appraisal of each method is given in "The Normal Skull" by Shapiro and Janzen.[13]

PROPOSED METHOD OF PINEAL LOCALIZATION

A new method of pineal localization on the lateral skull roentgenogram is proposed. This consists essentially of deriving a point

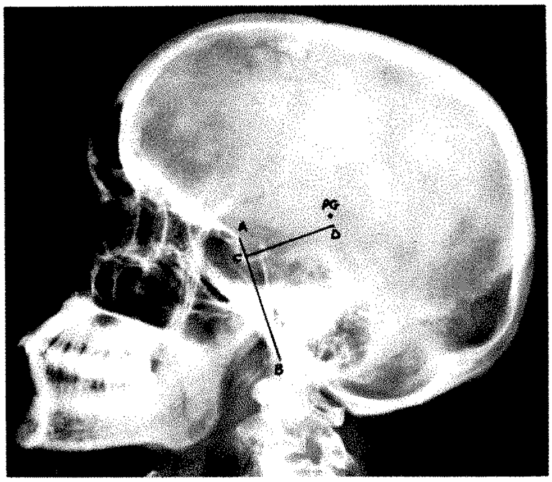

Fig. 3. New Method of Pineal Localization. A line (AB) connects the tuberculum sellae and the anterior lip of the foramen magnum (basion), and a perpendicular arm (CD) is erected 1 cm. from A. The expected position of the pineal gland is given by the point D, 5 cm. from C.

which represents the expected position of the pineal body, and it is based on reference to the basilar portion of the cranium (Fig. 3). The expected position should correspond to the actual position of the pineal gland or should not deviate from it to any significant extent. These considerations make the technique radically different from previously described methods.

Two reference points need be identified; namely, the tuberculum sellae and the caudal tip of the basi-occiput which is in effect the anterior edge of the foramen magnum (basion). A line is drawn joining these points and a perpendicular arm is erected to this line 1 cm. from the tuberculum. The expected position of the pineal body is given by a point 5 cm. on this arm. This method can be simplified by the use of a transparent celluloid guide.

ASSESSMENT OF ACCURACY OF THE NEW METHOD

A total of 100 adults without any history of pre-existing intracranial lesions was examined to compare the accuracy of the new method with previous methods. The latter included the Vastine-Kinney method, the proportional method of Fray and the cranio-angle method.

Table I

CASE MATERIAL STUDIED

	Male	Female
Chinese	30	22
Indian	14	10
Malay	14	7
Others	2	1
Total	60	40

The subjects under study were principally drawn from three racial streams which comprise our local population. A breakdown into the various ethnic groups is given in Table I.

The mixed composition of the sample is an advantage in skull work from the anthropologic viewpoint. However the study group consisted largely of brachycephalic skulls (Table II). This predominance of brachycephaly is indeed a characteristic of the Chinese and the Malay, whereas the third racial component, the Indian, shows a tendency to mesocephaly.

Roentgenograms were taken with the Schönander skull table, using a fixed focus-film distance of 28 inches (70 cm.). The latter detail is important as it has a bearing on the new technique of pineal orientation and will be discussed later. A true lateral view is a prerequisite for successful identification of the various reference points.

The calcified pineal gland was readily visualized in all cases under consideration. The calcification was seen as a single nodule, or as an irregular agglomeration of calcium or in the form of a shell. In all these types, the approximate center point of the calcification was used for the purpose of measurement and analysis of results.

Table II

TYPE OF SKULLS IN STUDY GROUP

Brachycephalic	70
Mesocephalic	22
Dolichocephalic	8
Total	100

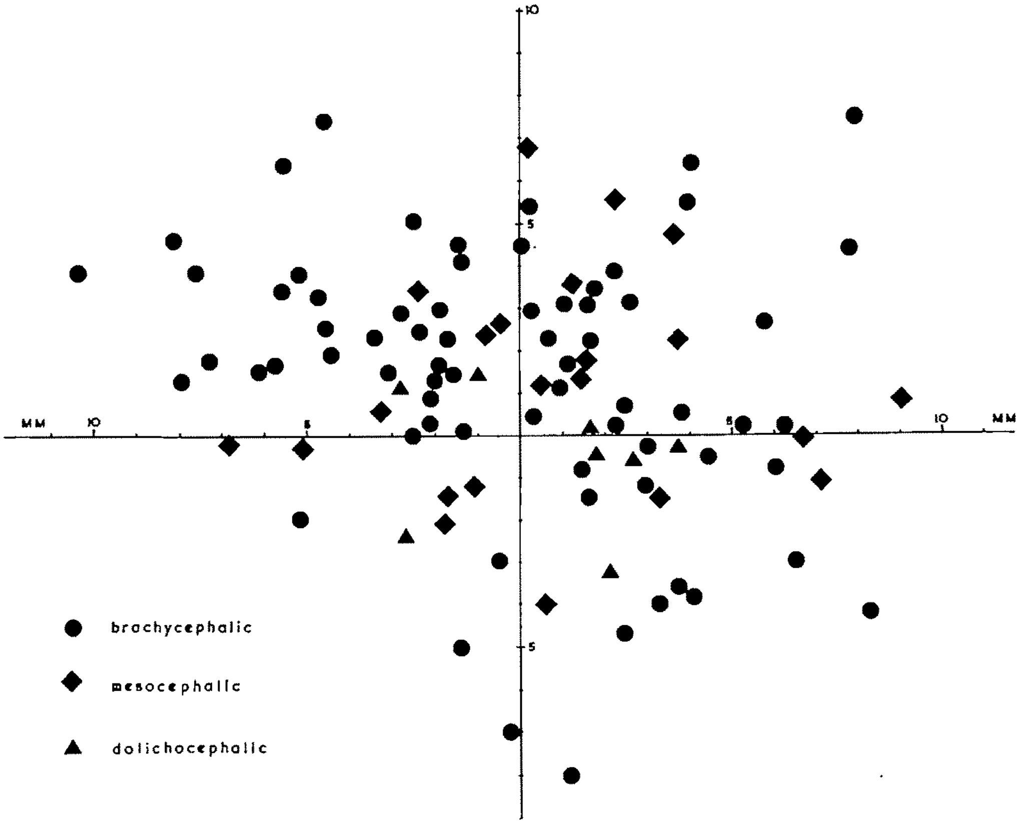

Fig. 4. Scatterogram of the result, using the new method of pineal localization in 100 normal cases. It gives the distance and direction of deviation in individual cases from their respective expected positions. The latter are represented by a common point formed by the intersection of the abscissa and the ordinate. The assessment of direction of deviation is made with reference to the nasion-tuberculum line, which is parallel in all cases to the abscissa. The pineal gland of each case is also typed with regard to skull shape.

Care was taken to differentiate the pineal calcification from the habenular calcification in view of their close proximity.[14,15] The appearance of the latter varies from a barely discernible fleck to a well-formed crescentic structure lying about 4 mm. anterior to the pineal gland. The reported incidence of visualization is approximately 30 per cent. Smith[14] showed that where habenular calcification was present, the pineal calcification was concurrently seen. The risk of a mistaken identity was therefore negligible.

RESULTS

The new method was found to have a reasonable degree of accuracy. In the trial series of 100 cases, the deviation of the expected position of the pineal gland from its actual position averaged only 5 mm. (Fig. 4). If a deviation of 10 mm. is accepted as the upper limit of the normal range, only 2 cases might be considered pathologically displaced, giving an accuracy of 98 per cent. The result of the investigation of this new method is graphically depicted. This shows the relation of the pineal gland in individual cases from their respective expected position and also gives an idea of the direction of deviation using the nasion-tuberculum axis as the reference base-line.

In contrast, the other methods employed were shown to be inadequate. The Vastine-Kinney method and the Fray proportional method had an accuracy of 78 per cent and 79 per cent in the anteroposterior direction and an accuracy of 88 per cent and 96 per

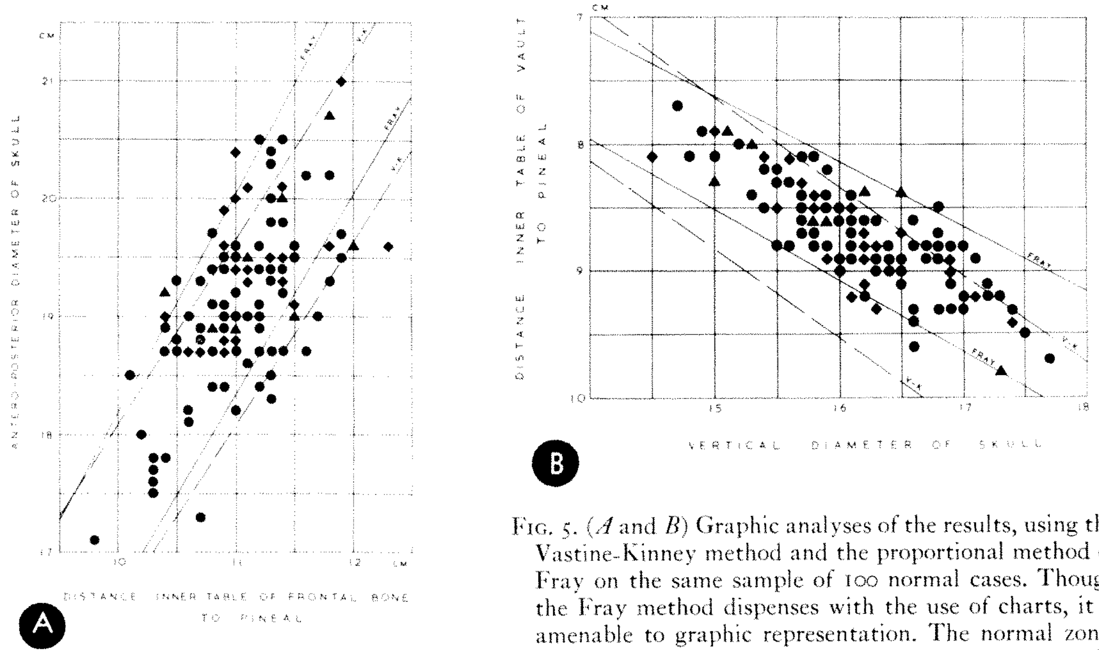

Fig. 5. (*A* and *B*) Graphic analyses of the results, using the Vastine-Kinney method and the proportional method of Fray on the same sample of 100 normal cases. Though the Fray method dispenses with the use of charts, it is amenable to graphic representation. The normal zones of these two particular methods are indicated on the charts and pineal glands falling outside the zones are considered pathologically displaced. The chart in *A* is used for the determination of anteroposterior pineal displacement. Here the distance of the pineal body (A of Fig. 1) is plotted against the sum of the distances of the pineal to the frontal and to the occipital bone (A+B). This sum is approximately equal to the greatest anteroposterior diameter of the skull. In *B*, used for the detection of vertical displacement, the measurement of the pineal body to the inner table of the vault (C) is plotted against the sum of the distances of the pineal to the vault and to the skull base (C+D). This sum is approximately equal to the vertical diameter of the skull.

cent in the vertical direction (Fig. 5, *A* and *B*). The cranio-angle method was found to be grossly inaccurate (Fig. 6, *A* and *B*). Poor identification of the anatomic reference points was, in the writer's opinion, a major contributory factor.

DISCUSSION

The results of this investigation have shown convincingly the superiority of the new technique to the other available methods of pineal localization used in the trial. The allowable range of normality used for the new method does not differ appreciably from that of the Vastine-Kinney and the Fray proportional method.

Despite the limited number of dolichocephalic and mesocephalic skulls in the study group, it would appear that the new method can be applied with equal sensitivity to skulls of such shapes. This does suggest that the basilar portion of the cranium which is used as the landmark of reference might be a function of the skull configuration. Thus the landmark might be used successfully with adaptation for the anatomic localization of other intracranial structures, particularly deep-seated midline ones.

The technique depends on the use of two other reference points on the cranium which should present no difficulty in identification. The tuberculum sellae is located at the top of the anterior wall of the pituitary fossa. It is a somewhat rounded structure, frequently superimposed by the bases of the anterior clinoid processes. The tuberculum is readily recognized except where the sulcus chiasmaticus produces a marked concave impression (Fig. 7g). In such a case, the new method would not be applicable, but such an occurrence is rare as shown by di Chiro and Lindgren.[2]

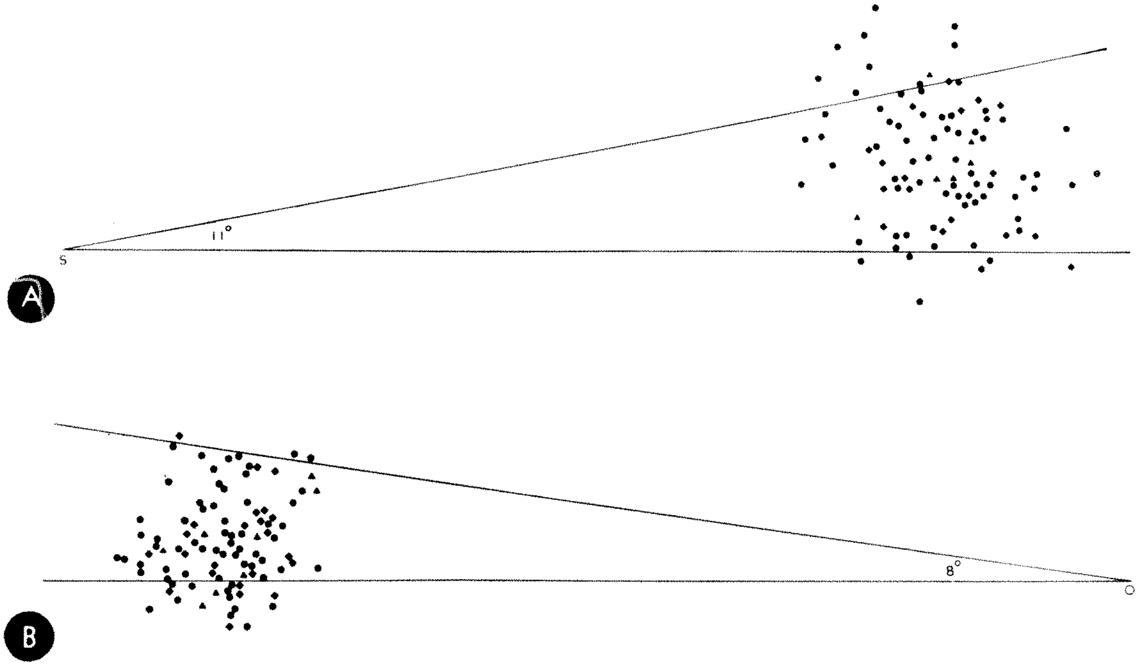

Fig. 6. (*A* and *B*) The results with the cranio-angle method of Fray, showing gross insensitivity of the method.

The tuberculum is little if at all affected in shape and position in the event of raised intracranial pressure. This is important as this pineal orientation method, like the others, is principally utilized in the localization of space-occupying lesions. Excluding intrasellar and parasellar lesions, an intracranial lesion first erodes the dorsum sellae, then the floor and subsequently the anterior wall of the pituitary fossa. The tuberculum is only affected in cases of gross intracranial pressure.[6,9,11]

A true lateral skull position and adequate roentgenographic penetration are essential for the visualization of the basi-occiput and the basion. The basi-occiput is seen as a tongue-like structure bounded by a cortical layer of bone with the basion at its caudal tip. As an additional guide, the basion is located about 5 mm. directly above the apex of the odontoid process.

Apart from the simplicity of technique, the new method has the added advantage that it is capable of modification to suit local condition and practice. This can be achieved by varying the site and the length of the perpendicular arm. This is necessary where the roentgenographic magnification is different. Again, adjustment may be required in order to take into account the predominance of a different skull configuration in a different locality.

SUMMARY

A new method of localizing the pineal gland on the lateral skull roentgenogram is

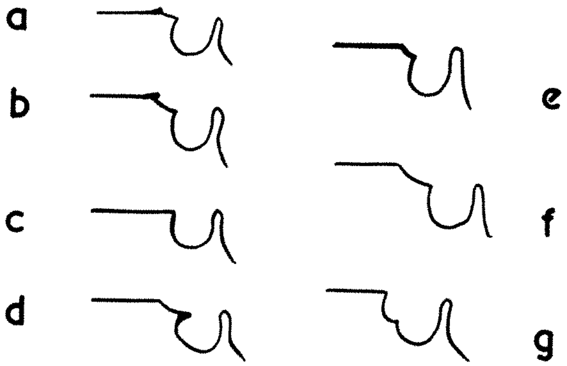

Fig. 7. Diagram showing the most common variations of configuration in the presellar region. The tuberculum sellae is the bony prominence at the top of the anterior border of the pituitary fossa. (Reproduced by permission of *Acta radiol.*[2])

proposed. The technique has the advantage of simplicity, and furthermore, can be adapted to suit local requirements and practice.

The method consists essentially of erecting a perpendicular arm to a line connecting the tuberculum sellae and the anterior lip of the foramen magnum. This arm is sited 1 cm. from the tuberculum and the expected position of the pineal gland is given by a point 5 cm. on the arm. Pineal glands in their normal situation should not deviate from this expected position by a distance exceeding 1 cm. In a sample of 100 normal cases, this method had a decided superiority in accuracy of pineal localization over some of the older methods. Though the sample consisted predominantly of brachycephalic skulls, it would appear that this method can be applied with equal sensitivity to skulls of other shapes.

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CALCIFICATION IN INTRACRANIAL METASTASES*

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IN RECENT years there have been several reports of calcification within metastases in the liver recognizable in the roentgenogram.[1–4] In such instances the calcification was generally seen in a necrotic area in a bulky metastatic deposit. In most of the cases, the primary neoplasm was in the colon. A primary tumor in the breast and a melanoma have also resulted in liver metastasis with calcification.

Cerebral metastases are common but reports of roentgenographic demonstration of calcification in these lesions are rather infrequent. A review was made of 136 cases which had autopsy proof of intracranial metastases in order to determine how often histologic examination showed calcium in the intracranial mestases. All of these cases had had skull roentgenograms taken and the findings on these films, or the radiologist's reports were reviewed.

FINDINGS ON SKULL ROENTGENOGRAMS

The findings are summarized in Table I. The most common abnormality reported was pineal displacement but this was not found as frequently as with primary brain tumors because the metastases were usually multiple and scattered in different areas of the brain. The displacements recorded include those from the midline as well as displacements upwards, downwards, forwards or backwards. Roentgenologic evidence of increased intracranial pressure was surprisingly rare, probably because the intracranial masses were increasing in size so rapidly that the skull changes did not have time to appear. Most of the cases were also examined by angiography or air studies but the results of these investigations have not been reviewed. Calcification within the metastases was detected prospectively by the radiologist in 2 cases (Fig. 1, *A* and *B*; and 2). The calcium deposits in both cases were similar, occurring as multiple small specks of increased density 0.5 to 2 mm. in diameter within an area of 1 to 3 cm.

Table I

FINDINGS ON PLAIN SKULL ROENTGENOGRAMS IN 136 PROVED CASES OF INTRACRANIAL METASTASES

Findings	No. of Cases
Normal	95
Pineal Displacement	21
Increased Pressure	14
Bone Metastases	9
Calcification in Tumor	2
Choroid Plexus Displacement	1

PATHOLOGIC FINDINGS

The most common site of the primary tumor was the lung (Table II). The distri-

Table II

PRIMARY SITE OF 136 PROVED CASES OF INTRACRANIAL METASTASES

Lung	66
Breast	17
Unknown	
Complete Postmortem Examination	6
Postmortem Examination of Head Only	10
Large Bowel	9
Melanoma	8
Kidney	4
Adrenal	3
Ovary	3
Thyroid	2
Osteogenic Sarcoma	2
Nasopharynx	2
Chorioepithelioma	1
Stomach	1
Fibrosarcoma of Thigh	1
Gallbladder	1

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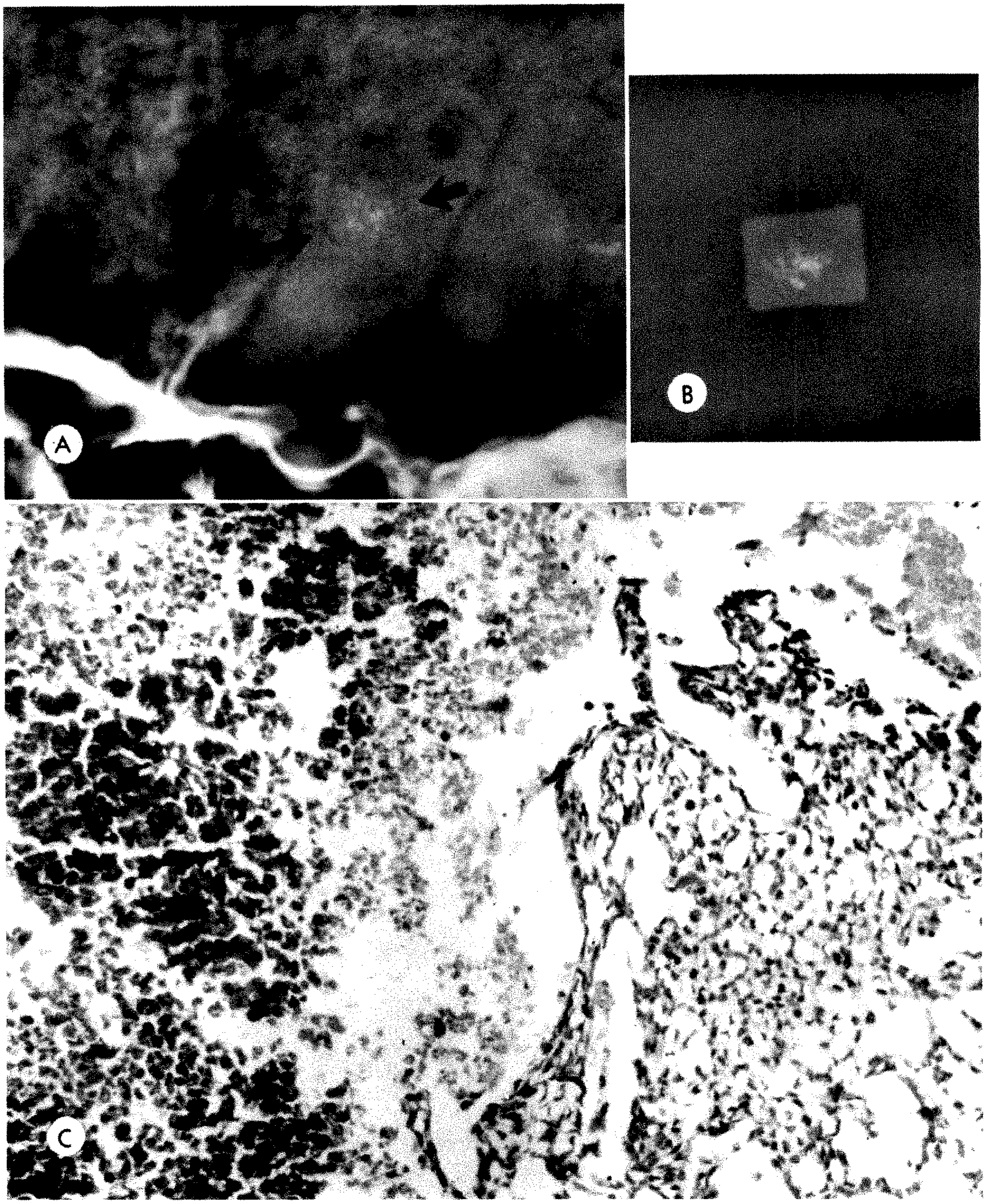

Fig. 1. (*A*) Admission lateral skull roentgenogram of a patient with carcinoma of the breast showing punctate calcification projected above the sella turcica (arrows). The pineal gland is also calcified and in normal position. (*B*) Roentgenogram of calcification of the autopsy specimen. The calcification proved to be in a large metastatic nodule in the left thalamus. (*C*) Photomicrograph of specimen shown in *B*. Amorphous darkly stained deposits of calcification are seen in one area of tumor necrosis.

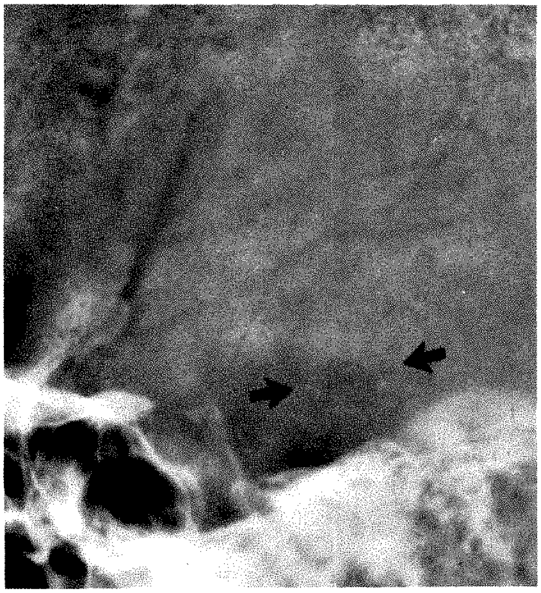

Fig. 2. Lateral skull roentgenogram of patient with carcinoma of lung showing punctate calcification posterior to the sella turcica (arrows). This was found at autopsy to be in a large metastatic nodule in the mid-brain.

bution of the intracranial metastases is shown in Table III. Most of the cases showed more than one intracranial metastasis at the time of death, and a number of the patients died within a few days of admission to the hospital. This suggests that by the time cerebral metastases are diagnosed, they are usually multiple. Of the 136 autopsy cases, 9 showed calcium within the intracranial metastatic tumor when examined microscopically. In every instance the calcium was found within masses of cellular debris in necrotic areas and occurred most often as coarsely granular or amorphous irregular deposits in the larger metastatic nodules in which necrosis was a prominent feature (Fig. 1*C*). In 1 case the calcium was in a metastasis in the globus pallidus and some or all of it may have been pre-existent. Of the 9 cases showing calcification, 5 were metastatic from the lung, 1 was metastatic from the breast, 1 was metastatic from the sigmoid colon, and in 2 cases the primary tumor was not determined because a complete autopsy was not performed. The microscopic appearance of the last 2, however, suggested an origin in the gastro-intestinal tract.

Table III

INTRACRANIAL SITES OF METASTASES IN 136 CASES

Cerebral Cortex and White Matter	100
Cerebellum	71
Basal Ganglia and Thalamus	19
Leptomeninges	38
Venous Sinuses	2
Corpus Callosum	3
Brain Stem	24
Pituitary Gland	9
Intraventricular	
Lateral Ventricle	1
Fourth Ventricle	3

SUMMARY

One hundred and thirty-six proved cases of intracranial metastases were reviewed. The most common site of the primary tumor was in the lung. Calcification was seen histologically in 8 of the 136 cases and detected prospectively on skull roentgenograms in only 2 instances.

As has been noted in metastases to other organs, the calcium occurred in areas of necrosis in the larger tumor nodules.

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THE INTERHEMISPHERIC SUBDURAL OR FALX HEMATOMA[*]

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SUBCUTANEOUS carotid arteriography is now a well accepted procedure for the immediate diagnosis of extracerebral hematomas in post-traumatic patients with progressive neurologic signs. In the majority of cases the subdural hematomas are located over the hemispheres.

A rather rare location of a subdural hematoma is the interhemispheric fissure. A search of the literature showed only a few such instances. Jacobsen,[3] in 1955, was the first to demonstrate an interhemispheric hematoma and emphasized, as did Brezina[1] and others[2,5,6,7] the importance of precise definition prior to surgery. With routine burr holes a hematoma along the falx would go unrecognized, especially when the neurologic symptoms did not indicate the exact topography of the lesion.

We have had the opportunity to diagnose an interhemispheric subdural hematoma in a patient who presented 5 years previously a grossly normal arteriogram and who was also studied postoperatively in order to follow the return of the callosomarginal artery to its normal course.

REPORT OF A CASE

P.N., No. 77329 BMCH. This 74 year old white male was admitted to our hospital for the third time in February, 1963. In a fall off a stool, he struck the back of his head and lost consciousness. The past history showed that the patient had had a subtotal gastrectomy for an ulcer in 1954.

The patient was first admitted in 1958 with left central facial weakness and slight impairment of the upper left extremity. The chest roentgenogram showed bilateral remote inflammatory disease. The electroencephalogram recorded low voltage activity on the right and then left. The right common carotid angiogram was reported as normal except for arteriosclerotic changes of the vessels (Fig. 1). The patient improved on anticoagulants and was discharged. He was placed on a daily regimen of bishydroxycoumarin.

The second admission in 1962 was for combined intracapsular cataract extraction. He remained on anticoagulants.

On the first evening of the third admission in 1963, the patient had a slight drift of the left upper extremity, but was alert and oriented. While in the hospital for observation, he continued to take regular anticoagulants against the advice of the doctor on service. Subsequently, he developed left hemiparesis, which progressed to hemiplegia of the left lower extremity, a left positive Babinski reflex, and minimal hypesthesia in the left lower leg. Vibratory sense was slightly decreased bilaterally.

Laboratory data were reported as normal, except for prolonged prothrombin time. Lumbar puncture showed an opening pressure of 156

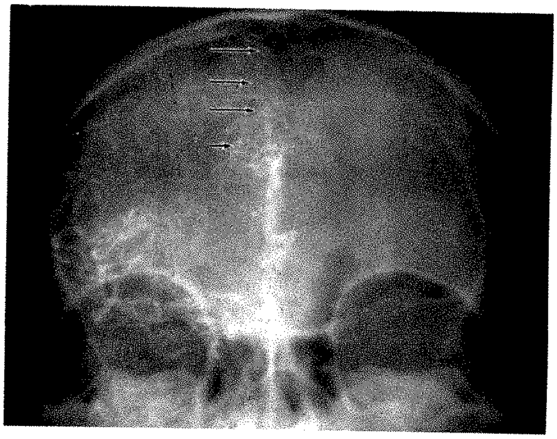

FIG. 1. Right carotid angiogram made in 1958, 5 years prior to the accident. The pericallosal and callosomarginal arteries are in a normal position (arrows).

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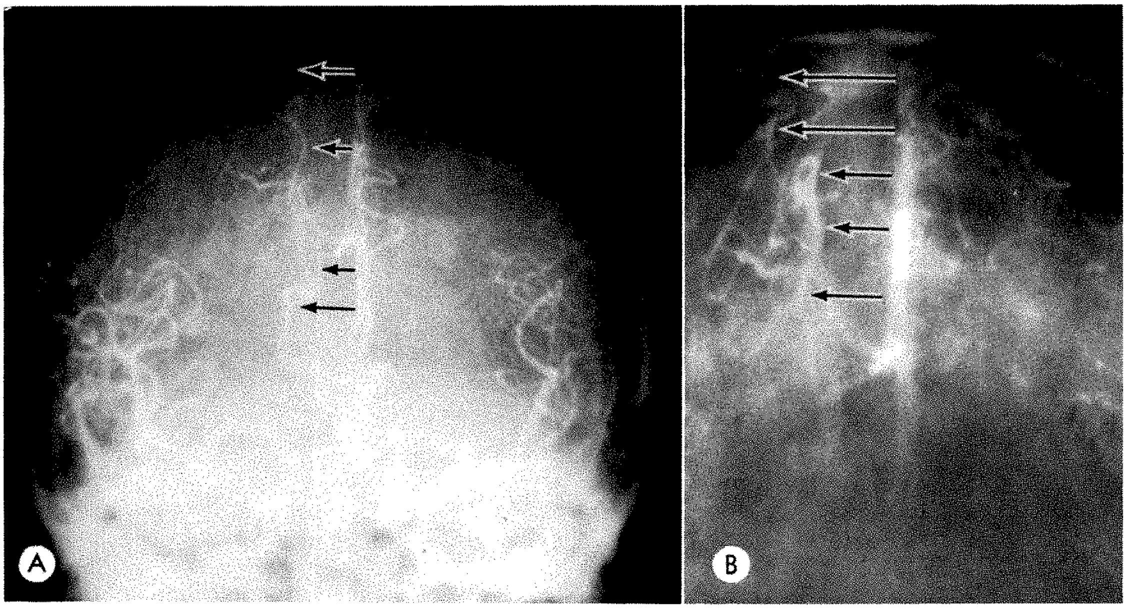

Fig. 2. (*A*) Right carotid arteriogram made in semi-Towne projection in February, 1963, 4 days after the accident, with compression of the left common carotid artery. Note the parallel separation of the anterior and middle internal branches of the callosomarginal artery from the pericallosal artery maintaining its normal course. An avascular zone is seen between the above named arteries. (*B*) Same study as in *A*. Close view of the displaced callosomarginal artery, still opacified in the late arterial phase, due to increased local intracranial pressure.

mm. H_2O. The cerebral spinal fluid appeared pinkish with a xantochromic supernatant and contained 2,225 red blood cells, 1 white blood cell, 64 mg. per cent protein and 65 mg. per cent sugar. No fracture was identified on plain roentgenograms of the skull.

Right common carotid arteriography was performed. As in most cases of subdural hematomas, the anteroposterior view proved to be the choice diagnostic study, demonstrating distinctly the crucial features which were as follows: (1) The pericallosal artery was opacified and in midline throughout its entire course in the cisterna of the corpus callosum. (2) The anterior and middle internal branches of the callosomarginal artery turned away from the midline and were displaced parallel to the pericallosal artery (Fig. 2, *A* and *B*). (3) A distinct avascular space was seen between the pericallosal and callosomarginal artery. Repeated injection with compression of the left common carotid artery emphasizes the above described findings (Fig. 2*B*). (4) The arterial and venous phases in the lateral view were compared and superimposed upon comparative roentgenograms made 5 years previously and demonstrated no significant diagnostic changes. (5) The internal cerebral vein was demonstrated in the normal anatomic position, without a shift.

The above findings were felt to be consistent with a subdural hematoma located to the right of the interhemispheric fissure. Because of a severe neurologic deficit, the patient was explored through right frontal and parietal parasagittal burr holes and approximately 40 cc. of blood was evacuated from the area. The postoperative course was one of slow continuous improvement. The left hemiparesis improved markedly and the patient was ambulatory at the time of discharge, prior to which a repeat right carotid arteriogram was obtained. The right callosomarginal artery returned to its normal relation to the pericallosal artery and the space void of vessels was no longer visible (Fig. 3). The patient regularly attends the clinics and continues to remain ambulatory.

DISCUSSION

Campbell and Campbell,[2] in 1961, described a left interhemispheric hematoma with identical findings, including the separation of the pericallosal and callosomarginal artery. However, in this case the hema-

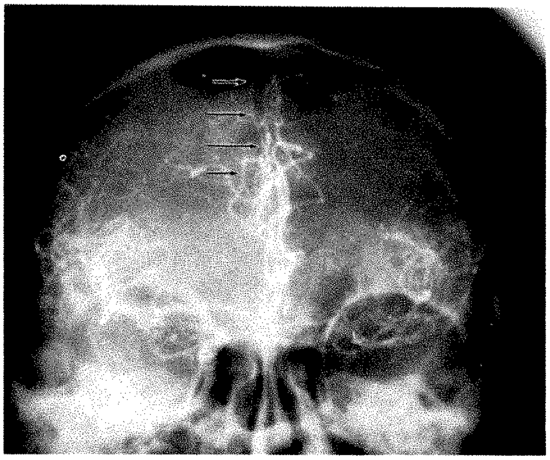

Fig. 3. Right postoperative carotid angiogram made in March, 1963, with contralateral compression. The anterior and middle internal branches of the callosomarginal artery have almost regained their normal position. The avascular space is no longer present (compare with Fig. 1).

toma spread over the convexity also, causing an additional shift of the anterior cerebral artery and internal cerebral vein. A hematoma which is isolated between the falx and medial surface of the brain, superior to the sulcus of the corpus callosum, usually will not shift the anterior cerebral artery or the pericallosal artery, and it does not affect the position of the internal cerebral vein. The vessel involved is the callosomarginal artery, which branches on the medial surface of the cerebral hemisphere. This artery and its branches are laterally displaced with the brain parenchyma by the collecting blood, thus creating the avascular space demonstrated in our patient. Jacobsen,[3] in 1955, also demonstrated an isolated interhemispheric hematoma with the typical avascular space. Our case is of particular interest because of its completeness. Pre-injury as well as pre- and postsurgical angiographic studies were available. The characteristic configuration of the hematoma is due to the lateral displacement of one or more branches of the callosomarginal artery. This can be visualized in the anteroposterior or semi-Towne views.

DIFFERENTIAL DIAGNOSIS

Kristiansen and Zimmer,[4] reported the cerebral angiogram of a 12 year old girl with an avascular interhemispherical lesion which resembled the arteriogram of our patient and also of the case reported by Jacobsen.[3] The girl had been treated for purulent frontal and ethmoidal sinusitis. Upon operation, a subdural interhemispheric abscess was noted which was removed. It is, therefore, evident that the patient's history and neurologic symptoms must be considered in the differential diagnosis.

SUMMARY

1. A case of an isolated interhemispheric subdural or falx hematoma is presented, which, to our knowledge, is unique in that normal pre-accident and postoperative angiograms are available for comparison.

2. The differential diagnosis is mentioned briefly.

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THE INCIDENCE OF ACQUIRED AND CONGENITAL FUSIONS IN THE CERVICAL SPINE*

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SEVERAL reports of fusion of cervical vertebrae have appeared in the literature.[1,2,4,5] Pan[3] found 8 "nonpathological" fusions in an unstated number of skeletons, 5 of which were fused at the level of the second and third cervical vertebrae (C 2–3). Other authors state that congenital fusions are "frequent" or "common."[1,2] However, there is no study of a large group of spines to determine the actual incidence of cervical spine fusion.

This is a report of the incidence of acquired and congenital fusion of two adjacent cervical vertebrae and the level in the column at which the fusion occurs. Skeletonized segments of the cervical spine were inspected and the observations correlated with their roentgenograms. The usefulness of the commonly employed roentgenologic diagnostic criteria was evaluated. The incidence of congenital fusion in clinical material also was determined.

MATERIALS AND METHODS

The cervical vertebral segments of 1,400 skeletonized specimens were studied.† These were from Caucasian and Negro cadavers ranging from 17 to 102 years of age at death. Those found to have two fused cervical vertebrae were examined in greater detail. The specific points of fusion of each specimen were recorded (posterior vertebral joints, uncovertebral joints of Luschka, laminae, or body of vertebrae), and note was made of the status of the involved intervertebral foramina.

After inspection of the skeletonized material, each specimen which had an osseous fusion was placed in the congenital or acquired category, using the criteria to be given later. Roentgenograms in posteroanterior, lateral, and both oblique projections were made of all specimens definitely showing congenital abnormalities and all specimens about which there was any doubt. To simulate the articulated condition, the spinal canals of the specimens were packed with cotton. Cardboard cassettes were used, and the technical factors were 70 kv., 50 ma., and 3 seconds at 40 inch target-film distance. The true incidence of congenital fusion was then determined by comparing the gross appearance with the roentgenograms, using the generally accepted roentgenologic criteria.[2] These criteria include absence or replacement of the disk by a line of increased density, anterior and posterior constriction at the junction of the fused vertebral bodies ("wasp-waist" appearance), smooth intervertebral foramina, or a single spinous process for 2 vertebral bodies. Another criterion is that the height of the block vertebra be equal to the height of 2 vertebral bodies minus the height of the intervening disk space.

RESULTS

Seventy-five fused segments were found in the 1,400 specimens studied. These occurred in 72 cadavers, 3 of which had 2 separate pairs of fused cervical vertebrae. Fusions were found in cadavers of 29 Caucasian males, 20 Caucasian females, 16 Negro males, and 10 Negro females. Age, race, and sex distribution of the material is shown in Table I.

The incidence of fusion of segments at the various levels is given in Table II. The figures in this table are for both congenital and acquired fusion. No case of atlanto-

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† From the Terry Skeletal Collection, Washington University School of Medicine, St. Louis, Missouri. The authors express their appreciation to Dr. Mildred Trotter, Professor of Anatomy, for permission to study this material.

* From the Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

TABLE I

AGE, RACE, AND SEX DISTRIBUTION OF 75 FUSED CERVICAL VERTEBRAL SEGMENTS

Cervical Level	No. of Fusions	Average Age	Caucasian Males	Caucasian Females	Negro Males	Negro Females
2-3	29	66.14	11	9	4	5
3-4	10	66.89	6	2	2	0
4-5	8	65.38	2	3	2	1
5-6	21	58.38	7	4	6	4
6-7	7	71.57	3	2	2	0
Total	75		29	20	16	10

TABLE II

INCIDENCE OF FUSIONS AT VARIOUS LEVELS IN 1,400 SKELETONIZED CERVICAL SPINES

Cervical Level	No. Fused	Per Cent of 1,400 Spines	Per Cent of 75 Fusions
1-2	0	0	0
2-3	29	2.07	38.8
3-4	10	0.72	13.3
4-5	8	0.57	10.7
5-6	21	1.50	28.0
6-7	7	0.50	9.3
Total	75	5.36	

TABLE III

INCIDENCE OF CONGENITAL FUSIONS IN 1,400 SKELETONIZED CERVICAL SPINES

Cervical Level	Congenital Fusions	Per Cent of 1,400 Spines	Per Cent of 10 Fusions
2-3	5	0.36	50
3-4	1	0.07	10
4-5	0	0	0
5-6	3	0.21	30
6-7	1	0.07	10
Total	10	0.71	

occipital or atlanto-axial fusion was found in this series. Twenty-nine fusions occurred at C 2-3, 10 at C 3-4, 8 at C 4-5, 21 at C 5-6, and 7 at C 6-7. Ten of the 75 fusions (0.71 per cent) were considered to be congenital (Fig. 1 through 4). Table III gives the levels of the congenital fusions. Five were at the C 2-3 level, 1 at C 3-4, 0 at C 4-5, 3 at C 5-6, and 1 at C 6-7. Bony fusion occurred between the bodies, laminae,

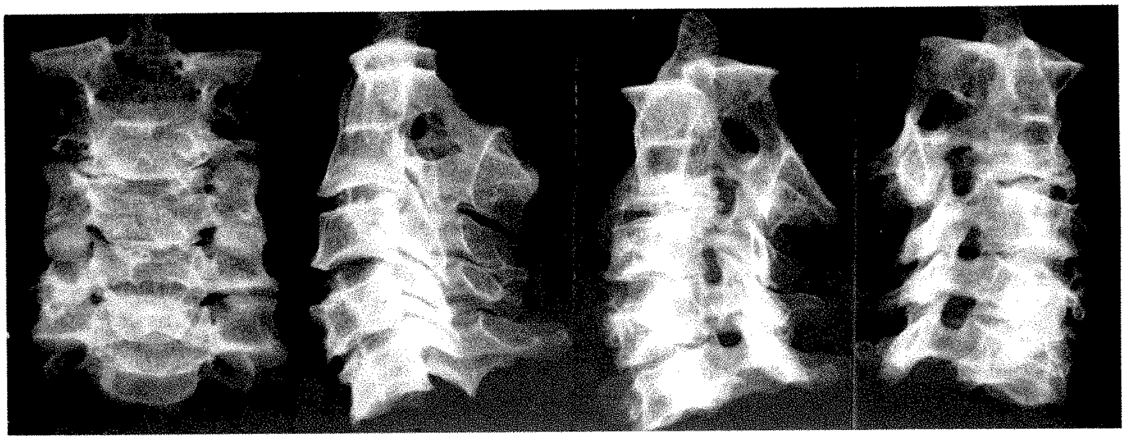

FIG. 1. Congenital fusion at C 2-3. There is a line of increased density and a slight constriction ("wasp-waist") where the disk space should be. The intervertebral foramina at this level are smooth and round. The laminae are fused posteriorly. (Spine 115 from the Terry Skeletal Collection.)

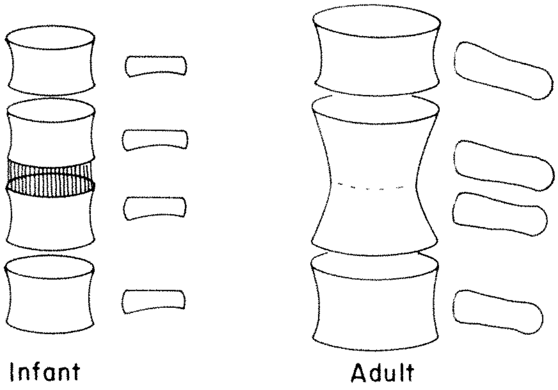

Fig. 2. Development of indentation at site of congenital fusion. Line drawing of lateral view.

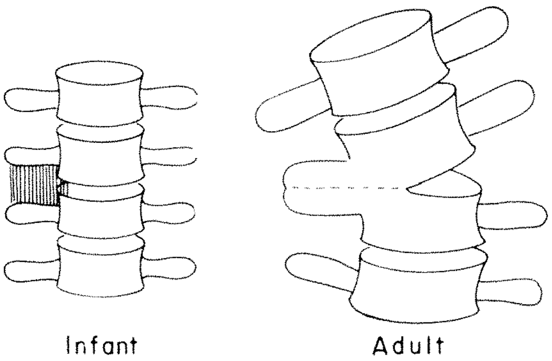

Fig. 4. Development of fusion and scoliosis as seen in Figure 3. Line drawing of anteroposterior view.

spinous processes, and posterior vertebral joints. Often the inferior fused lamina was thinner and more delicate than the superior lamina, and it was sometimes incomplete posteriorly. As a basis for comparing the clinical incidence of congenital fusions with the skeletonized material, all of the cervical spine examinations done at the Mallinckrodt Institute of Radiology during 1963 were reviewed. Of the 1,158 examinations performed, 7 (0.60 per cent) were diagnosed as congenital fusions. Four were at the C 2–3 level, and 1 each at C 3–4, C 4–5, and C 5–6.

The incidence of acquired fusions in the skeletonized material was 4.65 per cent. No attempt was made to attach a specific etiologic diagnosis to each of the acquired fusions. Bony fusion in the acquired cases was found to occur between the laminae, posterior vertebral joints, uncovertebral joints, and the bodies of the adjacent segments. It was often difficult to appreciate that a bony fusion was present on a roentgenogram of a specimen (Fig. 5). Encroachment on the involved intervertebral foramina by osteophyte formation was common in the acquired fusions. These data are summarized in Table IV.

DISCUSSION

Both congenital and acquired fusions are most frequent at the C 2–3 level and next

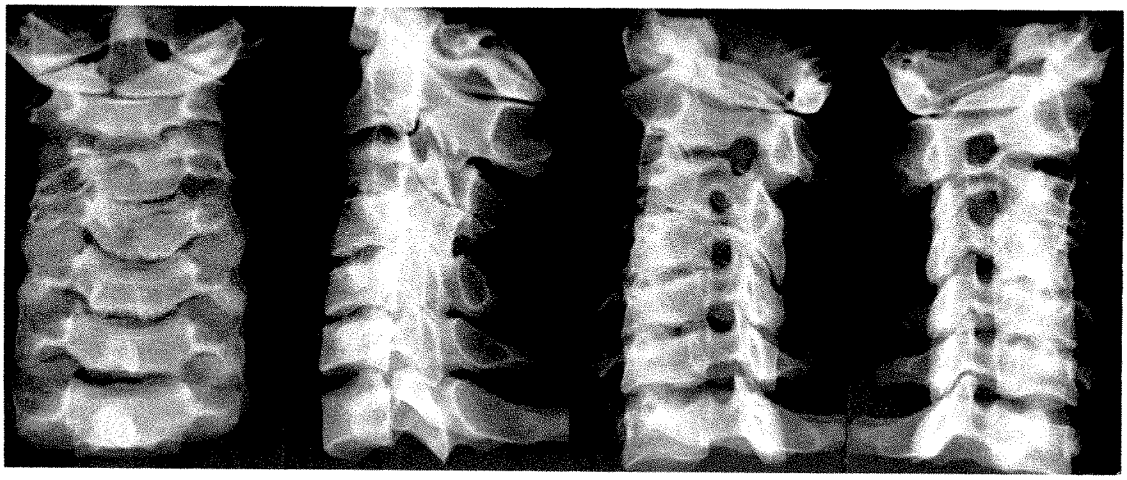

Fig. 3. Congenital fusion of lateral masses and lateral portion of bodies on left side. There is a slight scoliosis at this level. (Spine 940 from the Terry Skeletal Collection.)

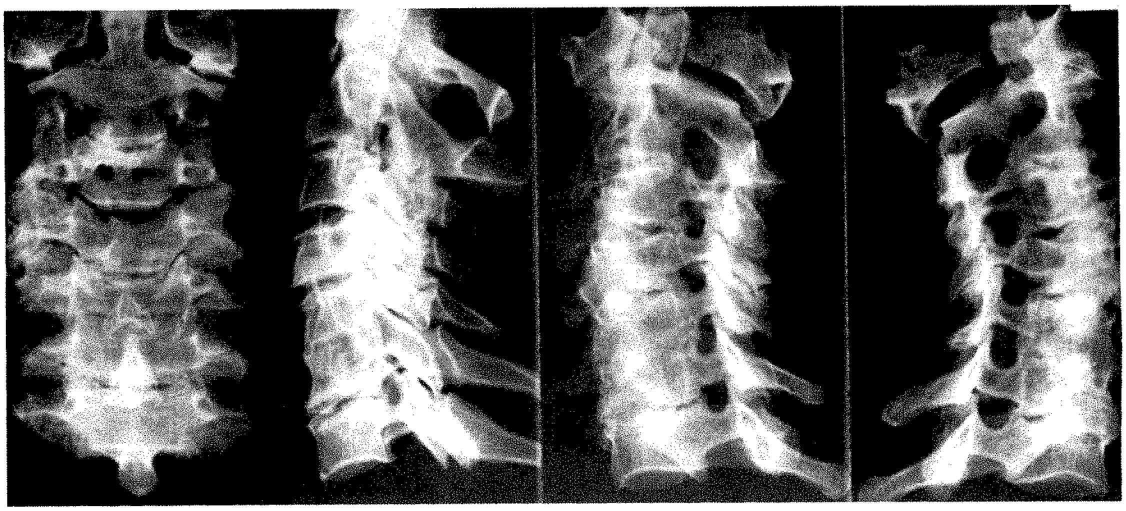

FIG. 5. Acquired fusion at C 5–6 and C 2–3. The fusion at C 2–3 might easily be missed on examination of the cervical spine in a living patient. There is spurring at the site of the intervertebral disk and encroachment on the intervertebral foramen which indicates that this is an acquired fusion. (Spine 1,125 from the Terry Skeletal Collection.)

most frequent at C 5–6. The incidence of congenital fusions, as determined from clinical roentgenograms (0.60 per cent), compares favorably with that found in the skeletonized material (0.71 per cent). On the other hand, the diagnosis of acquired fusion in clinical material probably does not approach the incidence of 4.65 per cent found in the skeletonized material.

In correlating the gross anatomic findings with the roentgenologic appearance to determine which fusions were congenital, the single most useful sign was the "wasp-waist" indentation at the site of the absent disk between the congenitally fused vertebral bodies. Figure 2 illustrates how the indentation could occur by fusion and cessation of growth at the indicated area and normal growth in the remainder of the vertebral bodies.

Another valuable sign of congenital fusion is absence of osteophyte formation at the uncovertebral joints. While several examples of acquired fusions with smooth intervertebral foramina were encountered, most of the acquired fusions had osteophyte encroachment there. Posterior bony bridging of the vertebral bodies with protrusion

TABLE IV

TYPE OF CERVICAL SEGMENT FUSION AND INCIDENCE OF ENCROACHMENT ON INTERVERTEBRAL FORAMINA IN 65 ACQUIRED FUSIONS OF CERVICAL VERTEBRAL SEGMENTS

Level of Fusion	No. of Spines	No. of Fusions at Each Location										Intervertebral Foramen Encroachment		
		Lamina			Posterior Vertebral Joints			Uncovertebral Joints (Luschka)			Body			
		R	L	Both	R	L	Both	R	L	Both		R	L	Both
2–3	24	1	9	6	1	8	14	1	1	0	7	0	5	4
3–4	9	0	2	2	2	2	5	0	0	0	3	3	2	4
4–5	8	2	1	3	2	1	4	0	1	0	5	1	0	6
5–6	18	1	0	1	2	0	3	0	0	1	14	0	1	9
6–7	6	0	2	2	0	1	2	0	0	0	7	1	1	3

into the spinal canal was seen only in the acquired fusions.

One spine was encountered which apparently had congenital fusion of the lateral masses and a portion of the bodies on one side (Fig. 3). Congenital fusion occurring as indicated in Figure 4 would explain this appearance.

SUMMARY

One thousand and four hundred skeletonized cervical spines were studied to determine the incidence of osseous fusions.

Fusions occurred in 5.36 per cent of the specimens. Of these, 0.71 per cent were congenitally fused and 4.65 per cent had acquired fusion.

For both acquired and congenital fusions the C 2–3 level was the most frequent and the C 5–6 level the next most frequent. In the acquired fusions, bony union was found to occur at the posterior vertebral joints, uncovertebral joints, laminae, and bodies.

The clinical roentgenograms of 1,158 cervical spines were reviewed, and congenital fusions were found in 0.60 per cent. Among the commonly used roentgenologic signs of congenital fusion, constriction at the junction of the fused vertebral bodies was found to be the most helpful.

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CERVICAL SPONDYLOTIC MYELOPATHY STUDIED BY AIR MYELOGRAPHY*

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IN SPONDYLOSIS deformans, degeneration of the disk occurs at multiple levels of the vertebral column, but when it is the cause of neurologic handicap, most often degeneration in the cervical spine is present. Since the extensive studies of Schmorl and Junghanns,[18] it has been known that progressive disk degeneration is common, that it may appear as early as the third decade, and that it affects practically everyone if sufficient age is reached. With this ubiquitous incidence and frequently asymptomatic state, it is necessary to look for special circumstances in affected patients to explain the neurologic defects. Our studies indicate that the significance of cervical spondylosis in relation to spinal cord disorders can be best appreciated with air myelography.

The use of air for myelography is not at all new, but its use has never been widespread in the United States. Dandy[7] in 1919 visualized the spinal cord by pneumoencephalography. Young and Scott[22] in 1937 introduced the first practical roentgenologic method. In 1939 Lindgren[9,10] elaborated on his technique with the use of warm moist oxygen and later laminagraphy. Murtagh and associates[13] reviewed experiences up to 1955 with particular reference to the cervical region and reported their use of air myelography as a general diagnostic method. In our viewpoint, pantopaque myelography is the best method for delineation of spinal cord tumors, lateral disk herniations and arteriovenous malformations; however, pantopaque myelography often does not meet the needs for a diagnosis of spondylotic myelopathy since it does not render the total picture needed in these cases. With modern equipment and new refinements in technique, the method now is more efficient and the examination directs attention to the status of the spinal cord, which is the proper object of concern in this disease.

TECHNIQUE

Our technique is basically that described by Lindgren.[10] Special roentgenographic equipment is absolutely essential since laminagrams must be obtained with the patient in the Trendelenburg position.

For puncture and roentgenography, the patient lies on his side in a 30–40 degree Trendelenburg position (Fig. 1). Care is taken to arrange the spine and head in such a manner that no lateral bending occurs; otherwise, the plane of a laminagraphic cut will not be in the spinal canal.

A suboccipital cisternal puncture is carefully made with the head in forward flexion. The needle is directed superiorly towards the deepest portion of the cisterna magna. The tip is "walked" over the foramen rim. The stylet of the needle is removed before penetrating the dura so that the flow of cerebrospinal fluid will indicate immediately when the subarachnoidal space has been entered. Oxygen is exchanged with cerebrospinal fluid in increments of 10 cc. When no more cerebrospinal fluid drips from the needle, additional oxygen is instilled to raise the intraspinal pressure to 200–300 mm. of water. This will distend the subarachnoidal space. Five laminagrams are obtained at 0.5 cm. intervals; one midline and two on either side of the midline. These are processed and viewed before proceeding to ensure adequate drainage and distention of the spinal canal. If the initial films are satisfactory, the needle is

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removed and the head and neck are extended backwards. The same layers of laminagraphic cuts are repeated.

Routine roentgenograms in the lateral projection of the thoracic and lumbar region are then made followed by laminagraphy of any suspicious areas. Our target-film distance is 60 inches.

ADVANTAGES OF AIR MYELOGRAPHY

The normal cervical spinal canal has a somewhat triangular shape in cross section with the smallest dimension being the sagittal diameter. Measurements on roentgenograms by various investigators[2,15,20,21] as well as our own studies show the sagittal diameter among normal persons to range from 12 to 22 mm., with an average of 17 mm. from C 3–4 to C 6–7 interspaces. The sagittal diameter of the cervical spinal cord is quite constant, averaging 9.5 mm. at C 3–4 and tapering slightly to 8 mm. at C 6–7 with variations of ± 1 mm.[12] The range of the available subarachnoid space in the sagittal plane in the remainder of the canal is from 2 to 8 mm. with an average of only 5.5 mm. Also, this subarachnoid space is wider on the ventral aspect of the spinal cord and measures 4 to 7 mm. with an average of 5 mm. On air myelograms, during flexion and extension movements of the cervical spinal column, the spinal cord remains in the same relationship to the canal walls, is always separated from the ventral surface of the canal by this buffer space and does not move forward or backward relative to the central axis of the canal.

The effects of cervical spondylosis, which have been observed to have a bearing on the spondylotic myelopathy, are brought out in air myelography by accurate delineation of the dorsal and ventral subarachnoid spaces as well as by accurate profile views of the spinal cord. Cervical spondylosis usually causes posterior interspace formations of either disk protrusions or transverse fibrocartilaginous bars, measuring 3–5 mm. in height, occasionally higher. There is commonly a kyphosis which may exaggerate the effects of these formations at the apex of the kyphotic curve. This reversed curvature is the factor which does not allow collection of pantopaque at the region of interest because one cannot "pool" the oil there. In many instances, there may be sclerosis of vertebrae, shortening of the vertebral bodies and an abnormal lordosis with shingling of laminae beneath each other, causing dorsal and ventral encroachment. The significance of these changes is in the spatial relationships of the canal and the demonstrable impingement on the spinal cord by the sum total of these changes.

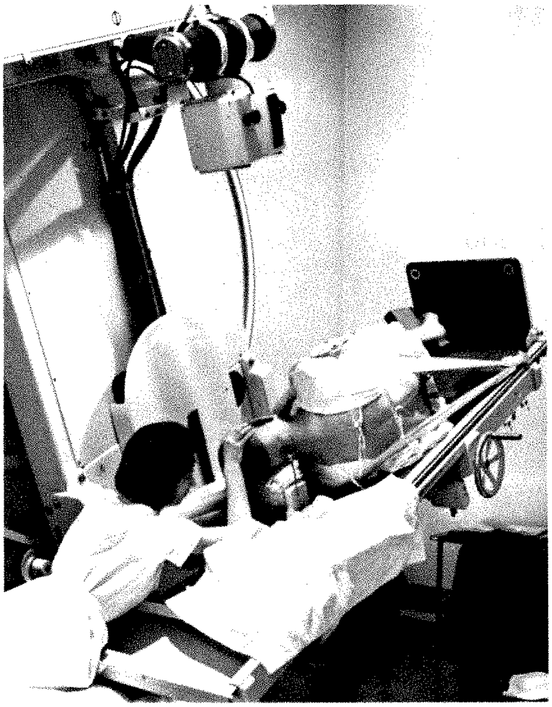

Fig. 1. Positioning and cisternal puncture accomplished in the Trendelenburg position with a technician ascertaining the straightness of the spine. Shoulder sling stabilizes the patient.

Pathologic dynamic actions of the cervical spinal column must also be considered in any case of spondylosis. These spatial relationships vary between phases of flexion and extension. There is increased bulging of the disk and a decreased sagittal diameter of the canal in positions of extension. Degenerated interspaces sometimes may be unstable, resulting in a "loosening" between the vertebral bodies. Penning,[16] Schmorl and Junghanns[18] and others have called attention to retrolisthesis resulting in cord compression between bony pincers formed by the anterior aspect of the arch of the inferior vertebra and the posteroinferior body of the superior vertebrae. Studies in full flexion and extension are made possible by air myelography without particular discomfort or hazard to the pätient who is in the lateral recumbent position.

The extent of involvement of the spinal cord by chronic disk degeneration can be best demonstrated by illustrations. Figure 2, *A* and *B* is a virtually normal study of a patient, but with mild, insignificant disk degeneration at C 4 through C 7. These laminagrams were chosen to show the protrusions which can appear on extension, but, in this instance, without encroachment on the cord. The posterior wall of the canal is thrown into folds in this position. Note also that the spinal cord maintains its position relative to the axis of the canal in both flexion and extension.

I. The usefulness of air myelography in cases of severe advanced spondylosis is illustrated by Case I.

CASE I. A 42 year old man had experienced progressive weakness and stiffness of the right arm and leg for about 9 months. He had noted

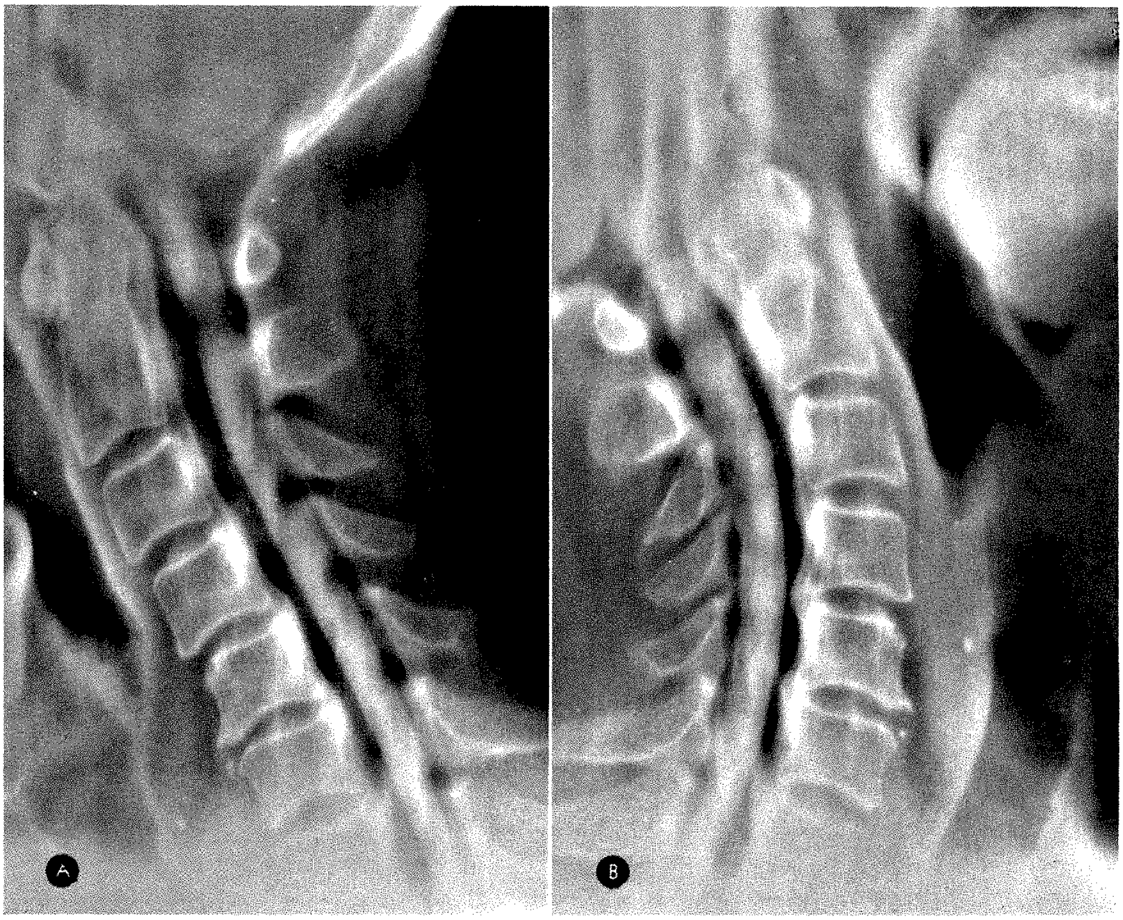

FIG. 2. (*A*) With forward flexion a wide ventral subarachnoid space is seen. (*B*) In extension, degeneration of the disk is evidenced by posterior protrusions. The ventral subarachnoid space is narrow but there is no significant encroachment on the spinal cord.

some wasting and flickering of muscles in the arms and shoulder girdles. Occasionally, pins and needles sensations were present in the forearm.

On examination, he was virtually confined to a wheel chair with a spastic right leg and arm which exhibited occasional clonic motions. He had a free range of motion of the neck. There was moderate wasting of the right deltoid, scapular, biceps, triceps, and intrinsic hand muscles. Tendon reflexes were extremely active on both sides with a bilateral Babinski sign. No objective sensory loss was found. Cerebrospinal fluid manometrics were clear and the protein was 44 mg. per 100 ml. Plain roentgenograms showed severe hypertrophic changes at C 2-3, C 3-4 and C 6-7 interspaces with some compromise of the neural foramina at all of these levels.

A pantopaque myelogram showed transverse bar defects at C 3-4 and C 4-5 interspaces (Fig. 3). Because of the ankylosis and limited mobility, an adequate cervical lordosis did not form and only a small amount of pantopaque could be pooled in the cervical region. Considerable amounts were scattered into the head when complete extension of the head and neck was attempted and found impossible to carry out. The entire canal could not be visualized adequately despite several different positionings.

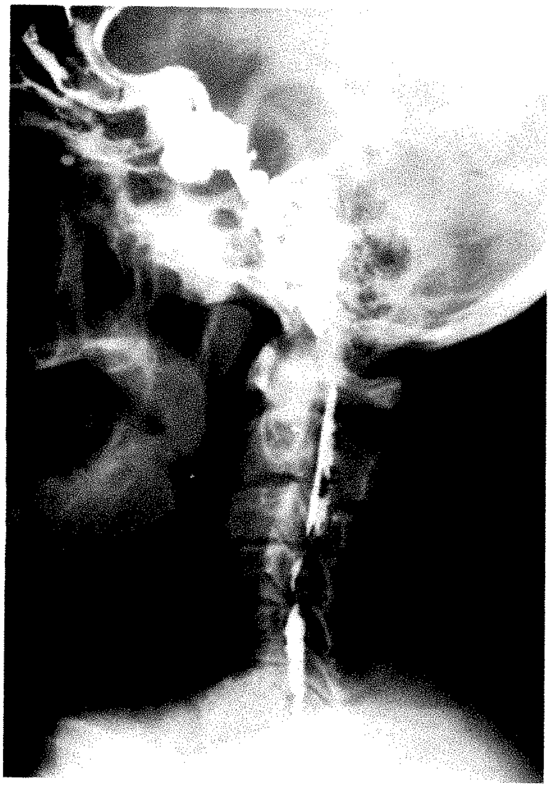

Fig. 3. Case 1. Pantopaque myelogram demonstrates a typical difficulty in "pooling" the oil in a spondylotic cervical spine.

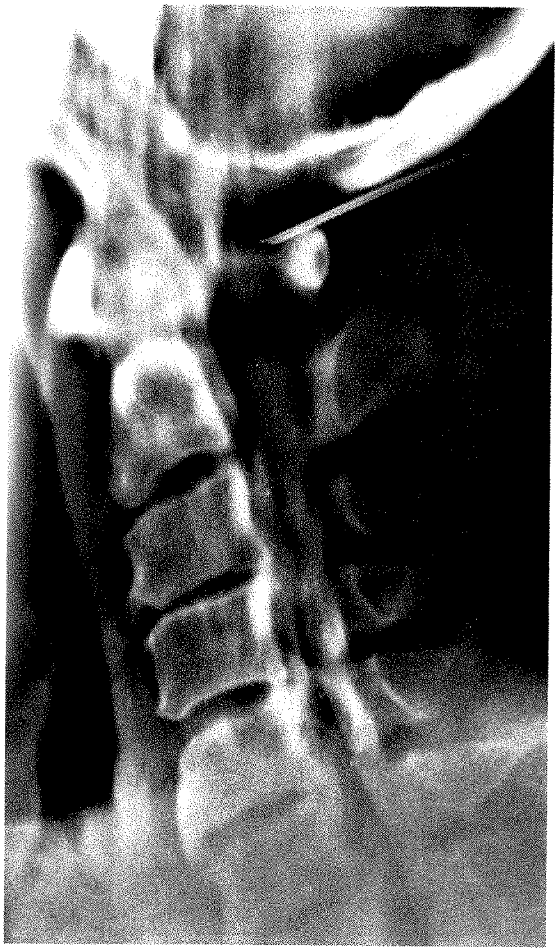

Fig. 4. Case 1. Corrugations of the cervical spinal cord secondary to considerable posterior protrusions and other soft tissue pathology in the floor of the cervical spinal canal are demonstrated. The dorsal subarachnoid space is not visible below C 2 and the ventral space is slit-like, or absent between C 2 and C 5.

Later, air myelography was performed. Not only were the defects caused by disk protrusions clearly shown but also the corrugated appearance of the cervical cord was visualized (Fig. 4). Cervical laminectomy was performed from the level of C 2 through C 6 and the severity of the demonstrated changes in the spinal cord at these levels was confirmed.

II. The intermediate stages of development of spinal cord encroachment have presented exceedingly interesting roentgenographic and clinical correlations. On air myelograms the disk protrusions occupy various proportions of the available subarachnoid space. In the intermediate group, there is a distinct air space ventral to the cord when the spine is flexed; however, on extension there is obliteration of the subarachnoid space dorsally and ventrally. Since, on extension, the air space becomes obliterated and the disk protrusions become more prominent, it is quite probable that further indentation of the spinal cord takes place.

Case II. A 41 year old white woman had had noticeable stiffness and pain in the neck for a period of 8 years. For about 1 month she had noted burning dysesthesias of the upper arms and shoulders and spasms of muscles of the upper back. Neurologic examination disclosed generalized weakness in both proximal and distal groups of the upper limbs, muscular tenderness and flexion contracture in the right arm. Bilateral atrophy of the deltoid muscles was present. The tendon reflexes were all very hyperactive with bilateral Hoffmann signs and a left Babinski sign.

Roentgenograms of the cervical spine showed flattening of the C 4–5 disk space with prominent spurring. At this level the sagittal diameter of the canal measured 12 mm. This narrowing in the midline was also confirmed by laminagraphy.

Laminagrams (Fig. 5, *A* and *B*) obtained in the lateral projection during air myelography showed bulging of the soft tissue portion of the disk space beyond the extent of the osteophytes at the level of C 4–5, C 5–6, and C 6–7 interspaces. An adequate ventral subarachnoid space was seen throughout, when the spine was flexed. On extension of the head, there was slight posterior subluxation of the C 4 vertebral body on C 5, causing a "pinching" of the cervical cord by the C 4–5 disk material.

III. In advanced stages of cervical spondylosis, changes occur that are due both to degeneration and to changes about the apophyseal joints. Some joints become totally fused by the osteophytes, whereas

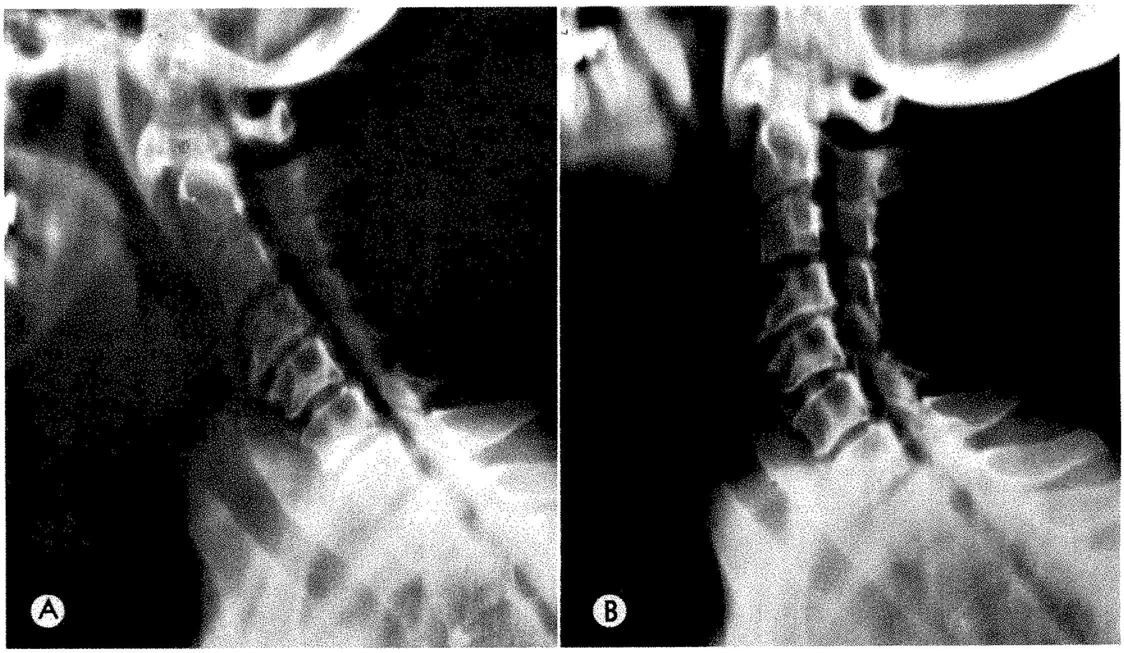

Fig. 5. Case II. (*A*) With flexion the ventral subarachnoid space is narrow but visible. The posterior projections from the interspace are also seen. (*B*) With extension there is slight retrolisthesis of the C 4 vertebral body and obliteration of the subarachnoid spaces by the compression.

the remaining joints show hypermobility. Pathologic subluxations readily occur because of ligamentous degeneration and abnormal stress when nearby segmental fixation occurs. Jones[8] has shown with cineroentgenography that when fixation occurs in the lower cervical region, compensatory hypermobility takes place in the remaining mobile upper cervical joints.

Case III. A 55 year old man presented himself for treatment with multiple disorders, the interrelationships of which were difficult to differentiate. He complained that for 2 years he had increasing loss of sensation in the right upper limb as well as spontaneous jerks of the legs and feet, especially at night. Five months previously, he developed temporary loss of vision on the right. Two weeks before admission he began to loose the vision of the left eye.

On physical examination he was plethoric, hypertensive (248/124), had clubbed digits, hepatomegaly, and dorsal kyphoscoliosis. Neurologically, he had spasticity in the right upper and both lower limbs, unsustained ankle and patellar clonus, a bilateral Babinski sign, loss of vibration sense on the right hand and left-sided analgesia below the C 5 dermatone.

Left visual acuity was 20/40. The disk was pale and edematous and small hemorrhages were noted. The right visual acuity was 20/80. The disk was pale and flat with gliosis and arteriolar narrowing.

Laboratory findings were hemoglobin 16.1 gm., hematocrit 50 per cent, blood volume 4,111 cc., cerebrospinal fluid protein 79 mg. per 100 ml. and an intraventricular conduction defect on the electrocardiogram.

Plain roentgenograms showed cervical spondylosis, especially evident between C 3-4, and on flexion and extension there was abnormal motion of these vertebral bodies. The sagittal diameter at this level was 9 mm. Arthritic changes in the intervertebral foramina and in the apophyseal joints at this level were evident.

Differential diagnosis included cervical spondylotic myelopathy, multiple sclerosis, and neurologic changes secondary to polycythemia vera or pulmonary disease. Ophthalmologic opinion was that the eye changes were hypertensive retinopathy with secondary optic atrophy.

Air myelography was performed with 110 cc. of oxygen (Fig. 6). There was marked encroachment on the cord anteriorly at the C 3-4 interspace due to posterior osteophytes as well as malalignment of the vertebrae. Air was seen posterior to the cervical cord at the level of the C 5 vertebral body but no air was seen anteriorly from C 3-4 through C 5-6 with either flexion or extension.

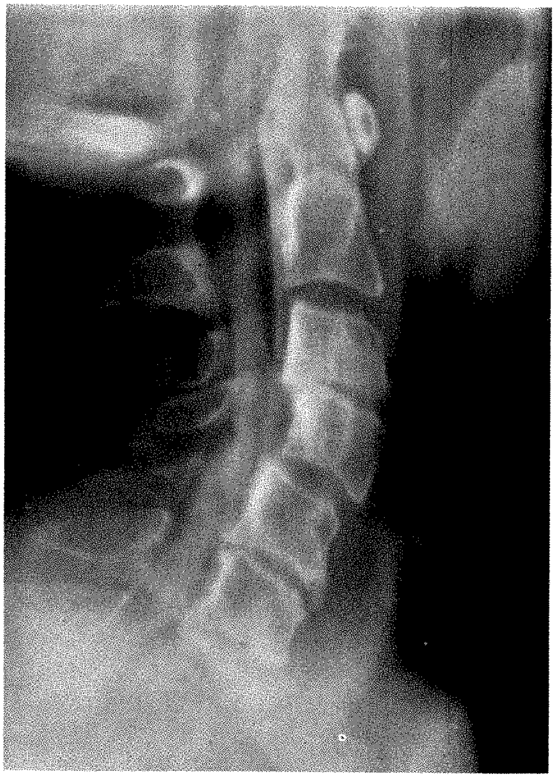

Fig. 6. Case III. Forward displacement of the vertebral body and lamina of C 4. The air myelogram shows a complete block below the level of C 3 body except for a small amount of air dorsal to the cord at C 5.

Anterior cervical disk excision and interbody fusion were performed at C 3-4 and C 4-5. The patient's subsequent course was improved with decreased spasticity and increased functional daily activities, but the sensory disturbances and evidence of spasticity of the right lower limb remained at the last follow-up examination 1 year after the operation. He also was treated with antihypertensive medications.

IV. Air myelography has proved to be a useful technique in evaluating postoperative cases of cervical spondylosis. When there is further progression of neurologic signs after surgical treatment, the various possible causes may be inadequate decompression, adhesive arachnoiditis, intrinsic cord degeneration or disk disease at other interspaces. When the previous operative intervention has been an opened dura and laminectomy, globule formation of pantopaque readily occurs in these areas. However, air will permeate around any adhesion and will permit myelography to be performed without concern for the possibility of aggravating chronic arachnoiditis due to iodine sensitivity, which must also be considered in the differential diagnosis.

Case iv. A 69 year old widow developed "burning cold" sensations in 1949 in the digits of the left hand, which in a period of 2 years advanced to the medial hand and forearm. The paresthesias were followed by numbness. In 1951, cervical spondylosis was diagnosed by pantopaque myelography, and at another hospital a cervical laminectomy at C 3 through C 6 was performed. No improvement ensued and 1 year later she developed the same progressive symptoms on the right which advanced to the level of the elbow by 1955. In 1960, she began to have difficulty in walking, and "burning cold" sensations on the lateral side of the left leg and foot. In 1962, there was numbness of the right toes as well as dragging of the right foot.

There was limitation of motion of the neck in all directions and a marked shock-like sensation with flexion of the neck. There was bilateral atrophy of the shoulder girdle muscles with prominent fasciculations.

The patient presented with a spastic gait and dragging of the right leg. There was spasticity and wrist clonus in the extensors and flexors. All of the deep tendon reflexes were abnormally brisk and accompanied by sustained ankle clonus and a bilateral Babinski sign. The sensory examination revealed dissociated sensory loss with marked analgesia bilaterally from C 4–C 8 dermatomes and mildly decreased touch perception in the hands, the left more than the right. There was severe loss of vibration perception in the lower limbs and hands, more so on the left than the right but without a distinct level.

Roentgenograms of the cervical spine showed the absence of the laminae of C 3 through C 6, posterior osteophyte formation and narrowing at all cervical interspaces without any prominence at any specific level. Moderate osteophyte formations impinged on the foramina at every level except the first. A lumbar puncture revealed normal pressure; manometric tests showed 32 mg. per 100 ml. total protein and a flat colloidal gold curve. An air myelogram (Fig. 7) demonstrated posterior osteophytes at the interspaces of C 3–4, C 4–5, C 6–7, but without any contact with the spinal cord. There was narrowing of the cervical spinal cord at the level of C 3–4 and below. At the lower margin of the laminectomy site, the cord appeared to be attached to the dura dorsally. It was concluded that central degeneration of the cord was present; whether it was primary or secondary was not clear. In any case, surgical treat-

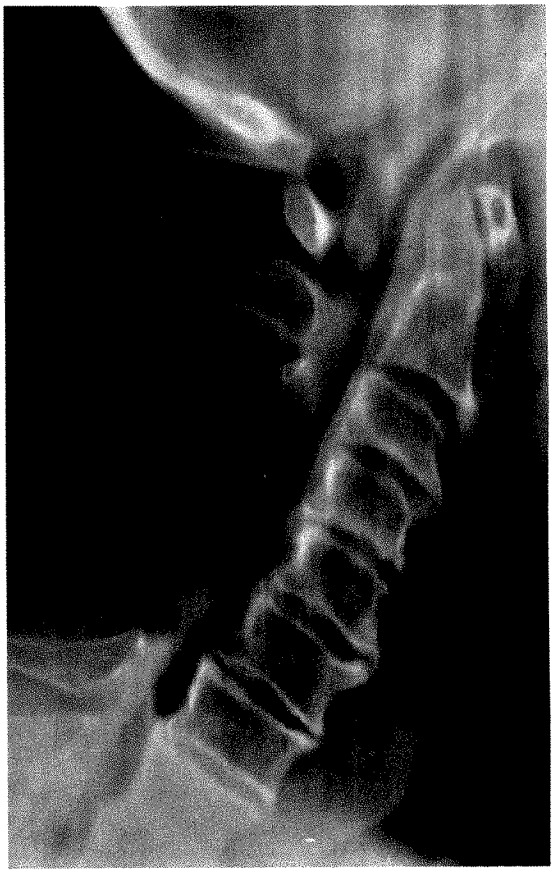

Fig. 7. Case iv. At the C 1 level the sagittal diameter of the spinal cord is 8.1 mm. At the C 4 level it measures 4.0 mm. Although there are cervical spondylitic changes at multiple levels, the ventral subarachnoid space throughout the cervical canal could always be seen in flexion and extension views.

ment was considered inadvisable since compression was evidently not a factor in this case.

DISCUSSION

Cervical spondylosis is now recognized as the most frequent cause of either spinal cord or nerve root deficits in elderly people.[14] As longevity increases, these disorders may be expected to increase correspondingly. Proper evaluation is necessary before treatment, particularly before operation is considered. In these spinal column disorders, there are evidently other factors than simple compression to be taken into account. Complex situations involving intermittent compression, entrapment effects, and angulation of neural structures are the result of deteriorated joints, deformities of curvatures, and unstable dynamic actions of the affected spine.

The normal elastic disk is held in place by strong ligaments and in effect distends the interspace pushing the vertebrae apart. With degeneration, the nucleus pulposus becomes desiccated, atrophied and lumpy and the ligaments become lax, bulging, and usually calcified. There can be nodular herniations, fibrocartilaginous bars in the canal floor and foraminal osteophytes. The vertebrae are also affected, sometimes by sclerotic changes with accompanying loss of height and by alterations in curvature. The most common deformity is kyphosis but excessive lordosis is also seen with shingled laminar arches and ligaments. Not all of the changes are significant or even symptomatic.

The manner and the circumstances under which these changes encroach upon the cord and nerve roots are of prime consideration in the evaluation of cervical spondylotic myelopathy. The cervical cord is the region of the central nervous system most exposed to flexion, extension, and other movements. It is normally surrounded by the buffer of a cerebrospinal fluid layer and is never directly in contact with the skeletal parts. Payne and Spillane[15] pointed out that in cervical spondylosis with myelopathy a high proportion of patients had a narrow sagittal diameter of the spinal canal, making the 3–5 mm. disk elevations of significance. Wolf, Khilnani and Malis[21] observed that the critical sagittal diameter at levels of degenerated disk disease was 11 mm.

The pathologic effects differ according to the position of the neck. Subluxations can occur in flexion or extension. Encroachment by the degenerated disk changes is greatest in extension. Trauma, which would not ordinarily be harmful, may produce neurologic deficits in the presence of spondylosis as in hyperextension,[19] in accidents, anesthetic intubations, or prolonged extension of the neck in pantopaque myelography.

Pantopaque myelography is often unsatisfactory because it is impossible to fill the kyphotic cervical canal adequately. The spinal canal profile is not accurately visualized. Part of the contrast material sometimes escapes into the intracranial cavity because of inability to extend the head and neck fully in these patients. The contrast medium is so dense that small nodular protrusions are sometimes missed in the lateral views. The medium in the lateral gutters on either side may mask midline defects.

From the clinical viewpoint, the most difficult problems have been, first, to obtain convincing evidence that the level of myelopathy corresponds to the spondylotic changes and, secondly, that the protean manifestations of spondylotic myelopathy are not confused with degenerative diseases,[3–5,11] such as amyotrophic lateral sclerosis, multiple sclerosis, subacute combined degeneration, syringomyelia, peripheral neuropathy, and other processes which may present concomitant but insignificant changes of chronic disk degeneration. A small proportion of patients with spondylotic myelopathy will have sufficient symptoms of painful compression of cervical nerve roots or certain shock-like paresthesias on flexion or extension movements such as to call attention at once to the cervical spine. A large proportion, however, have painless neck motions. Neurologic syndromes attributable to cervical spondylosis may consist of various combinations of purely motor system involvement, anterolateral tract involvement, lateral column syndrome and even transverse myelopathy. Even the lower motor neurone changes are not sharply limited to the exact level of compression but evidently extend above and below the level for some distance, presumably because of stretch injury of the cord.

In any case, the differential diagnosis between cervical spondylotic myelopathy and various degenerative diseases affecting the spinal cord is difficult. Air myelograms have proved useful by showing compression of the spinal cord, on the one hand, and atrophy of the cord, on the other.

Once the diagnosis of compression has been established, surgical treatment should be considered since the disabilities will invariably progress. Early treatment is important as the neurologic defects in spondylotic myelopathy are not always reversible and may only be arrested. Two surgical approaches are currently in use: (1) laminectomy, and (2) anterior cervical disk excision and interbody fusion. We prefer to use anterior cervical disk excision for large ventral protrusions at one or two interspaces and laminectomy for multiple interspace involvements. In certain instances, we have combined both operations. Air myelography accurately delineates the exact number of interspaces in which cord compression occurs. With the use of flexion and extension, this method determines when the cord is affected by abnormal motion and determines the need for stabilization. In addition, the dorsal aspect of the spinal subarachnoid space can be evaluated and the indentations of the dura, ligaments or laminae are readily visualized.

SUMMARY

Spondylotic changes in the cervical and lower thoracic spinal column sometimes affect the spinal cord by compression, by entrapment in a narrow deformed canal, or by unstable joint movements. Air myelography permits visualization of the spinal cord outline which is measurable and its position can be determined in views of flexion and extension. Since all parts of the spinal subarachnoid spaces are distended with oxygen, the spatial relationships in the sagittal planes between the sizes of the protrusions, spinal cord diameters and available subarachnoid space can be evaluated. It is particularly suitable when the usual differential diagnosis includes various neurologic disorders which often produce atrophy of the entire cord. The changes seen in the air myelograms may offer a rationale for the type of surgical treatment when compression or unstable joint action are the principal mechanisms of involvement.

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TUMORS OF THE SPINE AND THEIR RELATIONSHIP TO THE INTERVERTEBRAL DISK

A Roentgenographic-Anatomic Correlation

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THE cartilaginous plate of the intervertebral disk like articular cartilage is extremely resistant to invasion or destruction by tumor. On the other hand, destruction of articular cartilage in pyogenic infections of joints is a clearly established fact, although the exact mechanism by which this occurs is not known.

Norman[4] pointed out in his clinical study of the paravertebral thoracic line that "tumors consistently produce destruction of the vertebrae with extension of the tumor into the paravertebral space, causing a segmental bulge of the paravertebral line without involvement of the intervertebral disk space. Infection, on the other hand, destroyed the vertebrae and produced paravertebral extension; but, in every instance, the roentgenogram showed disk space narrowing or involvement."

The study of the paraspinal line emphasized the importance of the integrity of the disk in differentiating tumor from infection. The present aspect of our work concerns the relationship of spinal tumors to the intervertebral disk.

ANATOMIC MATERIAL

Sixteen anatomic specimens of the vertebral column extensively involved with either primary or metastatic tumor were studied. Information concerning the age, sex, primary tumor site, extent of involvement of the spine on the roentgenogram and the histologic diagnosis is presented in Table I. Special attention is given to the relationship of the tumor to the intervertebral disk.

Detailed microscopic examinations of the vertebrae, intervertebral disk, cartilaginous plate and adjacent soft tissue were made. In all but one instance, histologic evidence of invasion or destruction of the intervertebral disk could not be established. A striking example of the above is Case 14. This patient had a primary carcinoma of the lung and metastasis to the 6th, 7th cervical and 1st thoracic vertebrae, as well as a sizable soft tissue mass. In spite of considerable paravertebral soft tissue extension and invasion of contiguous vertebrae, the intervening disk spaces were uninvolved (Fig. 1 and 2).

The only exception was Case 6, a patient with a primary carcinoma of the stomach. The anatomic specimen demonstrated metastasis throughout the thoracic and lumbar spine with almost total collapse of several vertebrae and tumor infiltrating the cartilaginous plate and the annulus fibrosus of the 1st thoracic disk.

Other examples, listed in Table I, illustrate the absence of tumor invasion of elements of the disk, even where the spongiosa and bony end plate of the vertebrae are completely destroyed. In spite of soft tissue tumor extension, the cartilaginous plate and elements of the disk are spared. The clinical or specimen roentgenogram in every instance showed integrity of the interspace (Fig. 3 and 4; and 5 and 6).

DISCUSSION

Although no study clearly establishes why cartilage or intervertebral disk tissue resists destruction or invasion by tumor, the absence of a blood supply in both tissues may be a factor.

Ferguson[3] in his studies on fetal and infant spines has shown that the embryonal

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Table I

Case No.	Age	Sex	Primary Tumor Site	Extent of Spine Involvement on Roentgenograms	Histologic Diagnosis	Tumor Involvement of Intervertebral Disk
1	89	F	Carcinoma of kidney	From 4th thoracic to 4th lumbar vertebrae; involvement of body, pedicles and spinous processes	Renal clear cell carcinoma	None
2	50	F	Melanoma of labial fold	Osteolytic metastasis to the vertebral bodies of T_{12} and L_4 and spinous process of T_8, T_9	Malignant melanoma	None
3	68	M	Carcinoma of prostate	Osteoblastic metastasis involving the bodies of L_4, L_5 and 1st sacral vertebrae	Well differentiated adenocarcinoma	None
4	43	M	Bronchogenic carcinoma of right lung	Osteoblastic lesions of the 12th thoracic, 1st and 2nd lumbar vertebrae	Poorly differentiated metastatic bronchogenic carcinoma	None
5	52	M	Carcinoma of right kidney	Osteolytic and osteoblastic lesions of lower thoracic and lumbar vertebrae	Renal clear cell carcinoma	None
6	42	F	Carcinoma of stomach	Osteolytic lesions in all thoracic and lumbar vertebrae; collapse of T_7, T_8, T_{10}, and L_4	Poorly differentiated adenocarcinoma	At T_1, tumor extended into the annulus fibrosus
7	50	M	Multiple myeloma	Osteolytic destruction of most of the vertebral column	Multiple myeloma	None
8	40	M	Malignant melanoma of left thigh	Osteolytic lesions in all lumbar and 1st and 2nd thoracic vertebrae	Highly pigmented melanoma	None
9	72	F	Carcinoma of right lung	Lytic lesions of T_{12} and all lumbar vertebrae	Metastatic adenocarcinoma	None
10	46	F	Carcinoma of left breast	Predominantly osteolytic metastasis to lower thoracic and all lumbar vertebrae	Adenocarcinoma	None
11	68	F	Carcinoma of breast	Osteolytic metastasis to lumbar vertebrae	Poorly differentiated adenocarcinoma	None
12	56	F	Leiomyosarcoma of uterus	Several osteolytic metastatic foci in 1st to 4th lumbar vertebrae	Leiomyosarcoma	None
13	65	F	Multiple myeloma of spine	Extensive destruction of lower thoracic and lumbar vertebrae	Multiple myeloma	None
14	69	M	Adenocarcinoma of right lung	Destructive lesions of both C_6 and C_7 with collapse of the body of C_7 and partial collapse of C_6	Metastatic adenocarcinoma	None
15	58	M	Carcinoma of prostate	Osteoblastic metastasis to the lower thoracic and lumbar vertebrae	Adenocarcinoma	None
16	45	F	Carcinoma of cervix uteri	Osteolytic metastasis to lower thoracic and lumbar vertebrae (L_2 almost completely destroyed)	Epidermoid carcinoma	None

disk has a vascular supply, which gradually involutes with progressive differentiation of the disk in the infant's spine and ultimately disappears with completion of growth at about the age of 25.

Since metastatic tumor embolization to disk or cartilage would ordinarily occur by the blood borne route, the absence of a blood supply to both these tissues makes it improbable for tumor seeding. Therefore, the primary focus of involvement would be the tissue neighboring the disk, namely, the vertebra or paravertebral soft tissue. Should tumor erode or implant in the intervertebral disk, the low metabolic turn-over of this tissue makes it improbable for a tumor nidus to survive.

Similarly, Scherbel and Gardner[9] point out that in the presence of blood borne infection to the spine, it too must lodge primarily in the vertebrae and involve the disk cartilage secondarily.

The present authors commented that the exact nature by which articular cartilage or disk is destroyed in pyogenic arthritis is not yet known. However, Curtiss and Klein,[2] insofar as *in vivo* processes can be deduced from *in vitro* experiments, state that "gross cartilage destruction in septic

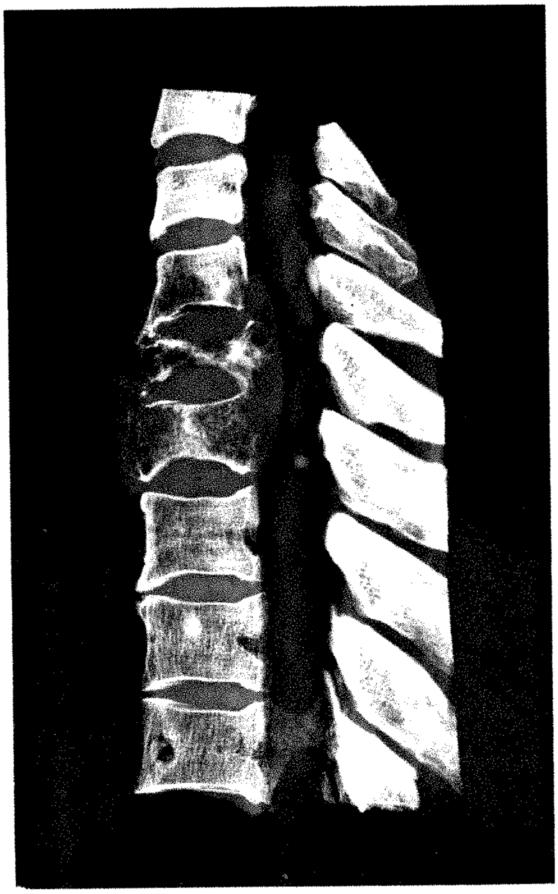

FIG. 1. Case 14. Roentgenogram of the anatomic specimen shows metastatic lung carcinoma involving C6 and 7, which is almost completely destroyed and collapsed, as well as destruction of T1. Tumor extends beyond the anterior and posterior confines of the vertebrae, producing a soft tissue mass without involvement of the intervertebral disk.

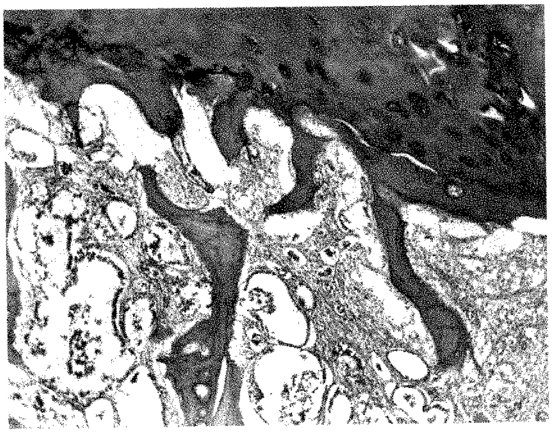

FIG. 2. Photomicrograph of the 7th cervical segment shows tumor extending to the cartilaginous plate of the intervertebral disk without invasion.

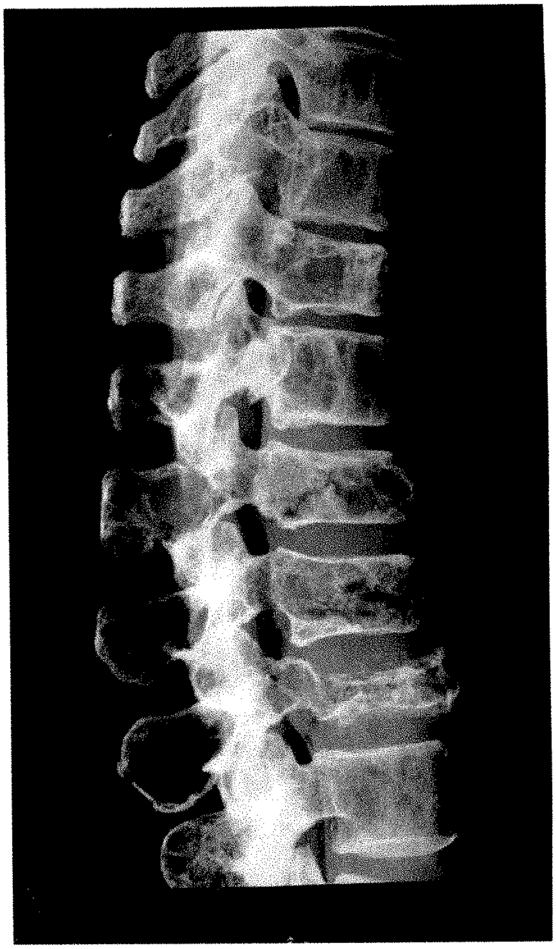

FIG. 3. Case 7. Roentgenogram of the anatomic specimen showing extensive osteolytic destruction of almost all the vertebral bodies, as well as the posterior neural elements by multiple myeloma. There is compression of several of the vertebrae. The intervertebral disk spaces are well maintained.

arthritis is not due to the action of leukocytic enzymes or plasmin on the cartilage matrix alone. The removal of collagen by enzymatic or mechanical means is necessary for cartilage destruction."

Tumor, by our present knowledge, does not elaborate an enzyme which can destroy collagen or attack the cartilage matrix. Furthermore, cartilage is an extremely durable tissue and refractory to mechanical erosion. Therefore, only rarely will one find destruction of cartilage or intervertebral disk by tumor.

Although Schinz *et al.*[6] report that "there are no known tumors of the intervertebral discs," and that in cases of vertebral tumor or vertebral metastasis the disk remains intact, Schmorl and Junghanns[7] make reference to a report by Schopper who commented: "invasion of the intervertebral disc is rare, but instances of disc involvement have been reported." Schmorl and Junghanns themselves had never observed a primary tumor of a disk or metastasis which involved the disk; however, they do say that the disk may be invaded by a primary or metastatic malignancy extending from neighboring structures.

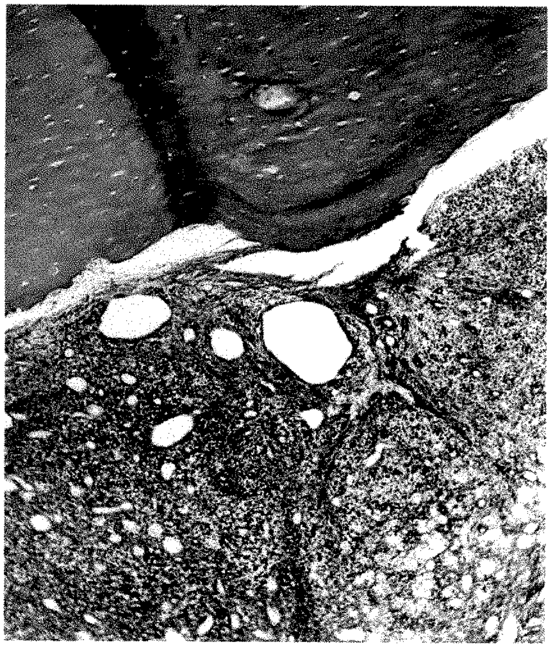

Fig. 4. Photomicrograph of a segment of the vertebra and the adjoining intervertebral disk shows extensive destruction of the vertebral body but no evidence of myeloma invading the disk.

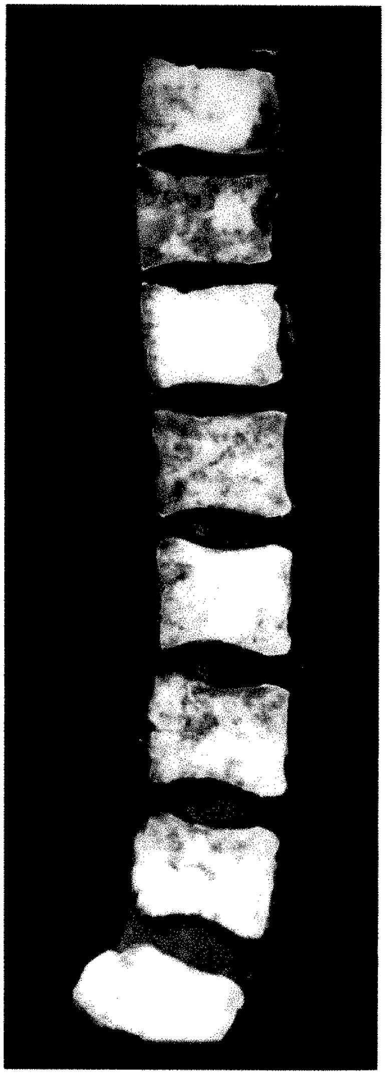

Fig. 5. Case 15. Roentgenogram of the anatomic specimen showing extensive osteoblastic metastasis of prostatic tumor to all the vertebral segments.

Recently, a patient operated upon for classic cervical disk symptoms was reported to us as having metastatic renal carcinoma to the intervertebral disk. This was confirmed by microscopic study. There was no involvement of the vertebrae. This example and the few reported in the literature, point out the possibility that tumor embolization to the disk may occur through persistent or unobliterated vascular channels.

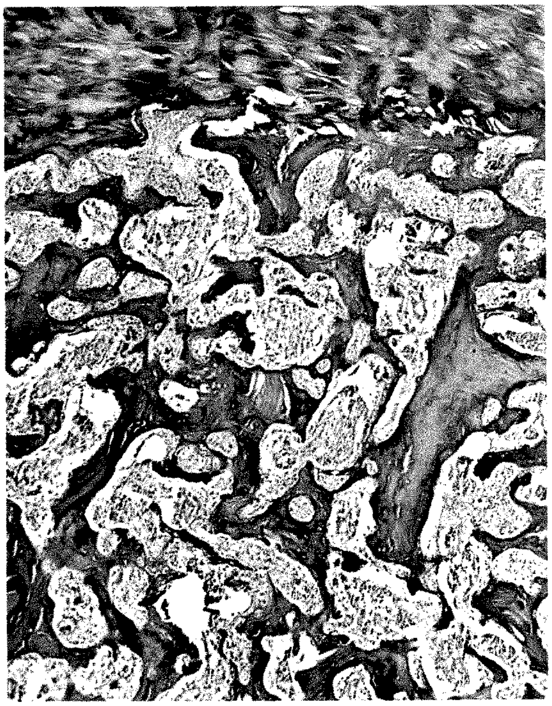

FIG. 6. Photomicrograph shows the marrow spaces extensively involved by tumor which extends to the intervertebral disk but does not invade this structure. A rather pronounced osteoblastic response to the tumor is noted in the vertebral body.

CONCLUSION

There are rare exceptions where primary or metastatic tumor will involve the disk and, conversely, a rare instance where infection destroys several vertebrae without invading the interspace. However, this anatomic study points out the consistency with which the intervertebral disk and the cartilaginous plate resist involvement by tumor. Therefore, it is reasonable to assume that if the roentgenogram shows destruction of the vertebral body with preservation of the disk space, tumor is present.

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THE PLACE OF DISKOGRAPHY IN RADIOLOGY AS BASED ON 2,320 CASES

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IN SPITE of the number of years since Lindblom[11,12] described diskography in 1944 and 1948, its acceptance has been limited even though there have been many conclusive studies in its favor.[1,2,3,7,10,15,16]

This hesitance is difficult to understand, for it is no revelation to acknowledge low back pain as one of the most common complaints physicians in all branches of medicine encounter and which often elude a positive etiologic diagnosis. This reluctance is also difficult to understand in this era of special procedures.

In view of the acknowledged need for an accurate diagnosis in cases of low back pain, a presentation of a summary of the results of lumbar diskography in 2,320 patients is warranted. The technique will be reviewed followed by the presentation of the results.

TECHNIQUE

The method is quite similar to that described by Lindblom.[11,12] Preliminary myelography is performed routinely for the purpose of excluding non disk pathology rather than for the purpose of delineating classic free fragments. Levels at which laminectomies have been performed are usually skipped. Either at the same sitting or on the following day, the disk spaces of the lower 3 to 4 levels are fluoroscopically identified, marked with an indelible skin marker, antiseptic is applied, the skin is draped, and procaine is injected intracutaneously and intramuscularly. The latter needle is used as a guide for the insertion of the spinal needle. For all but the very obese, a 3½ inch No. 20 spinal needle is inserted at each level up to the anterior arachnoid at the midpoint of the disk space. For the beginner an anteroposterior spot roentgenogram and a Polaroid roentgenogram in lateral projection should be taken for accurate localization prior to the insertion of a 5 inch No. 25 needle to the midpoint of the disk *per se*. The needle should not be used as a rongeur but inserted gently. When the disk needle penetrates the annulus, the sensation is comparable to the apt description of penetrating American brick cheese.[7]

Formerly, each disk was injected and roentgenographed in turn. At present 3 or 4 disks are injected in succession and roentgenographed thereafter in order to avoid prolongation of discomfort for the patient.

I believe that the most important point to stress is that rather than to follow an empirical rule for the volume of contrast material to be injected one should inject up to the point of resistance or reflux. If the latter does not occur, a maximum of 2 cc. is used. If the injection is properly performed, reproduction of pain in a magnified sense is to be anticipated in a pathologic disk. One can produce pain even in normal disks if the volume of contrast material is more than the normal nucleus will accept, with resultant extravasation. The latter may extend anteriorly causing abdominal or epigastric pain. If the extension is to the lateral longitudinal ligaments, atypical eccentric back pain referral is not unusual. Sometimes, with overenthusiastic injection pressure, posterior passage of contrast material through the annulus to the epidural space can occur in an atypical fashion. Fortunately, these defects can be identified as being artefactual by the sharp linear tracts surrounding the needle. These do not cause a problem in interpretation to the experienced diskographer.

The postinjection study includes anteroposterior spot roentgenograms, oblique spot roentgenograms to identify lateralized tracts if needed as an adjunct, and an across the table lateral roentgenogram.

TABLE I

DISTRIBUTION OF INTERVERTEBRAL DISK INVOLVEMENT IN 2,320 PATIENTS

Level	No. of Cases	Per Cent of Total
Single disk space involvement—846 cases		
L5–S1	568	24.4
L4–5	217	9.3
L3–4	39	1.6
Transitional	22	0.9
Two disk space involvement— 857 cases		
L4–5, L5–S1	678	29.2
L3–4, L5–S1	87	3.7
L3–4, L4–5	62	2.6
L5–S1, transitional	30	1.2
Three disk space involvement—380 cases		
L3–4, L4–5, L5–S1	380	16.3
Four disk space involvement— 32 cases		
L2–S1 or L3–transitional	32	1.3
Negative diskography	205	8.8

RESULTS

At the time of writing, 2,320 patients have had diskographic examinations at Mt. Sinai Hospital. The distribution of the findings is listed in Table I. As one might anticipate, the most frequently involved levels were L4–5 and L5–S1, together comprising 29.2 per cent of the cases. The second most frequent involvement was the single interspace of L5–S1, comprising 24.4 per cent of the cases. The complete breakdown of the distribution is shown in Table I.

Of the 2,320 patients examined since 1955 when the author began this series, which does not include the original cases reported from this institution by Friedman and Goldner,[7] 6,784 disk spaces were injected. The discrepancy of the number injected not being a simple multiple of the number of patients is due to the fact that those levels which had been disrupted by laminectomies were not injected, frequently spondylolisthesis levels could not be entered, and some patients had 4 levels injected. The small number of normals is due to the fact that the bulk of our patients have been selected primarily because their clinical pictures were abnormal but not classic for free fragment disk disease.

The distribution of the findings and responses to the injection of 6,784 disks are listed in Table II. Some pain response is listed in normal disks. This, in part, may be related to the volume of contrast material injected early in the series. It is probably worth emphasizing at this point that exclusive of local disk site pain at any one of the involved levels, inguinal distribution of pain is not uncommon at L2–3 or L3–4. Of course, gluteal, hip, and lower extremity pain referral is quite common at L4–5 and L5–S1.

In the series, 1,786 patients underwent surgery with the remarkably high level of roentgen-surgical correlation of 99.2 per cent or 1,783 cases.

INTERPRETATION

A fact which cannot be overemphasized is that the early discrediting of diskography was due to the inability to determine what constituted a normal disk. There is and was no difficulty in interpretation of the anatomically smoothly elliptical nucleus, with or without a sharp central translucent vacuum as seen at L3–4 in Figures 1*B*, 2 and 3. The problem involves the misinterpretation of varying degrees of degeneration in the form of decrease in the

TABLE II

PAIN RESPONSE IN VARIOUS DISK TYPES IN THE 6,784 DISK INJECTIONS

Type of Disk Configuration	Pain	No Pain
Normal disk without degeneration	32	176
Normal disk with some degeneration	73	357
"Impending" disk (form of normal)	78	265
Abnormal disk with annulus bulge	2,491	301
Abnormal disk with posterior epidural leak	3,011	—

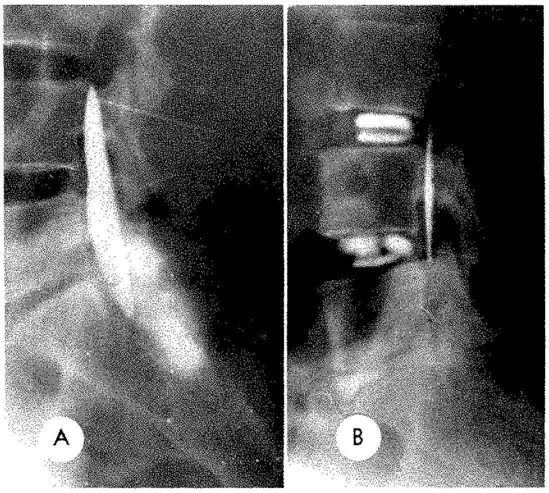

Fig. 1. (*A*) Lateral lumbosacral myelogram showing normal L5-S1 arachnoid oil column with narrow disk space. (*B*) Lateral diskogram of same patient showing normal L3-4 disk, normal but fissured L4-5, and flattened, fissured, as well as anterior and posterior extension of nucleus substance with posterior epidural leak.

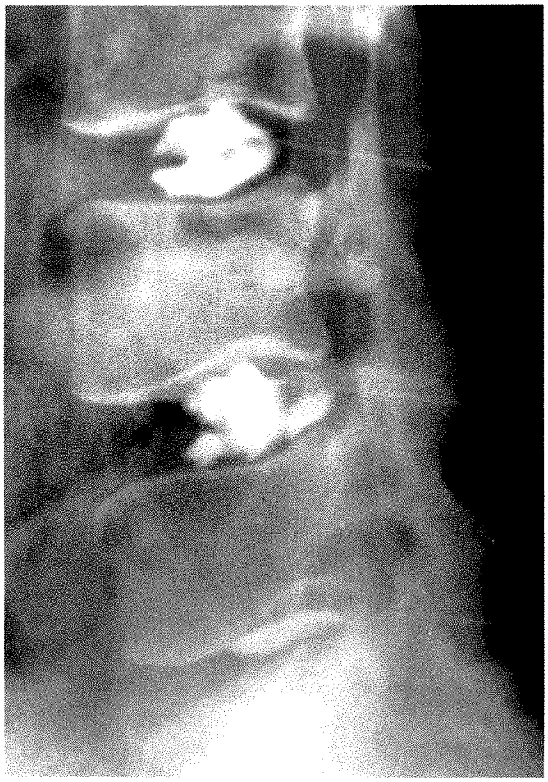

Fig. 2. Normal L3-4 disk, "impending" normal L4-5 disk, and flattened as well as fissured L5-S1 which is not pathologic in view of the absence of extension beyond the annulus confines.

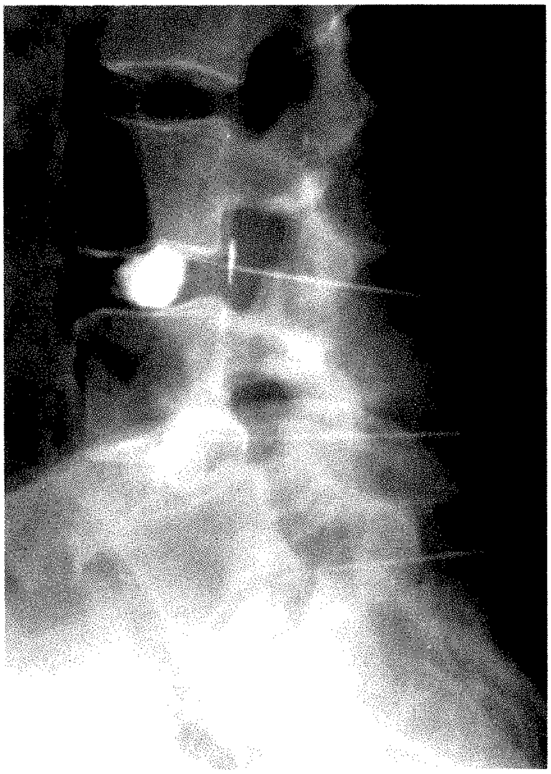

Fig. 3. Normal L3-4, fissured L4-5 with posterior annulus bulge. In addition, there is fill of a Schmorl node in the posterior inferior aspect of L4. The L5-S1 disk shows fissuring as well as loss of normal architecture. In spite of this, no posterior extension is present. Here too, a Schmorl defect is filled at L5.

vertical height of nucleus substance, fissuring, bilateral or anterior extension without actual extension beyond or significant bulging of the annulus (Fig. 1*B* at L4-5, Fig. 4 at L3-4 and Fig. 5*A*). These degenerative changes are not pathologic, but they have been improperly interpreted as such by the inexperienced in the field, thus resulting in poor roentgen-surgical correlation.

An intermediate disk configuration should be mentioned even though it is still within the limits of normal. For want of a better term, this is classified as an impending prolapse (Fig. 2 at L4-5). In this case the nucleus extends posteriorly to the annulus without bulging of the latter or without disruption of the latter.

The pathologic findings can be classified

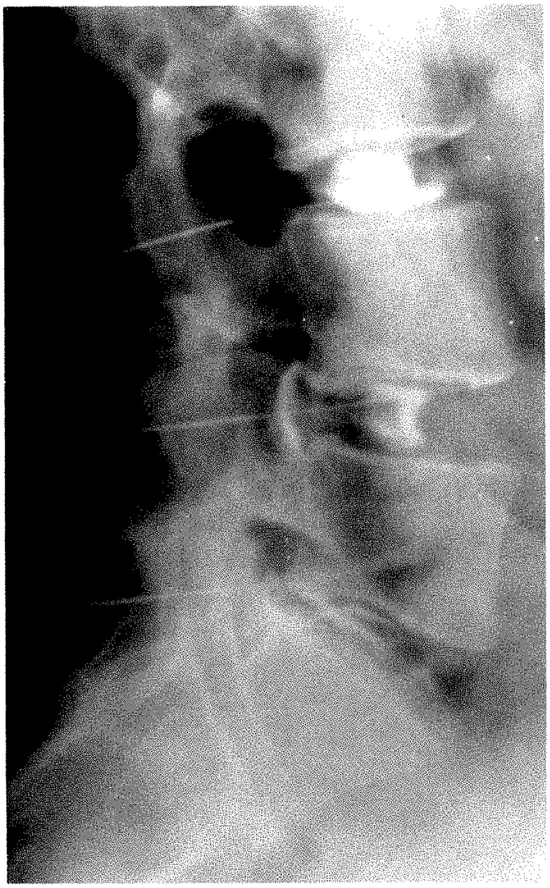

FIG. 4. L3-4 is fissured but normal, L4-5 maintains its vertical height but shows considerable posterior bulge. L5-S1 is flattened, fissured and extends posteriorly into the epidural space through an annulus disruption.

into those which have posterior annulus bulge without disruption of the annulus or epidural leak (Fig. 3 and 4 at L4-5). Simultaneous fill of Schmorl nodes is apparent in Figure 3. The second anatomic pathologic finding is the obvious posterior extension into the epidural space through a disrupted annulus (Fig. 1*B* at L5-S1, Fig. 4 at L5-S1 and Fig. 5*B* at L5-S1).

A separate category of pathologic disks is included. These are in the minority but have assumed more importance now that anterior fusions are being done.[8,9] These are the cases which may or may not have posterior protrusion but which show extensive fissuring as well as extension of nucleus substance in a markedly gross fashion to the point of elevating anterior or lateral longitudinal ligaments in spite of proper needle placement and small volume of injection of water soluble contrast material, *i.e.*, 2 cc. or less (Fig. 5, *A* and *B* at L4-5 and L5-S1).

DISCUSSION

Diskography was introduced in 1944 when dynamic angiography was coming into the forefront. As a result, the visualization of a static anatomic landmark did not evoke much interest among the more energetic "Surgical School of Radiology." At the same time, others legitimately feared its potential hazard to the annulus.

Neither attitude is considered justified by the author, for by virtue of its static character the disk can be quite positively categorized as enumerated in the Section describing Interpretation. This is in contrast to the more fleeting character of vascular opacification.

Likewise, when properly performed, traumatic annulus tear does not occur.[1,2]

The highest percentage of clinical disk pathology is found at the lower lumbar levels where myelography is more likely to give false negative results.[13] The greatest space between the posterior aspect of the vertebral bodies and the arachnoid occurs at these levels where significant defects can be missed on myelograms. This accounts for the various reports of myelographic accuracy varying between 65-98 per cent. It is at the lower 3 to 4 interspaces that diskography finds its niche. (Although lumbar diskography is considered worth the effort, the author has discontinued cervical diskography as being of doubtful value.)

Exclusive of the time and skill factors, its disfavor can be traced to what various neurologists, neurosurgeons, orthopedic surgeons, and radiologists label as significant disk pathology. Those who require the roentgen and surgical demonstration of a free fragment as the only criterion for call-

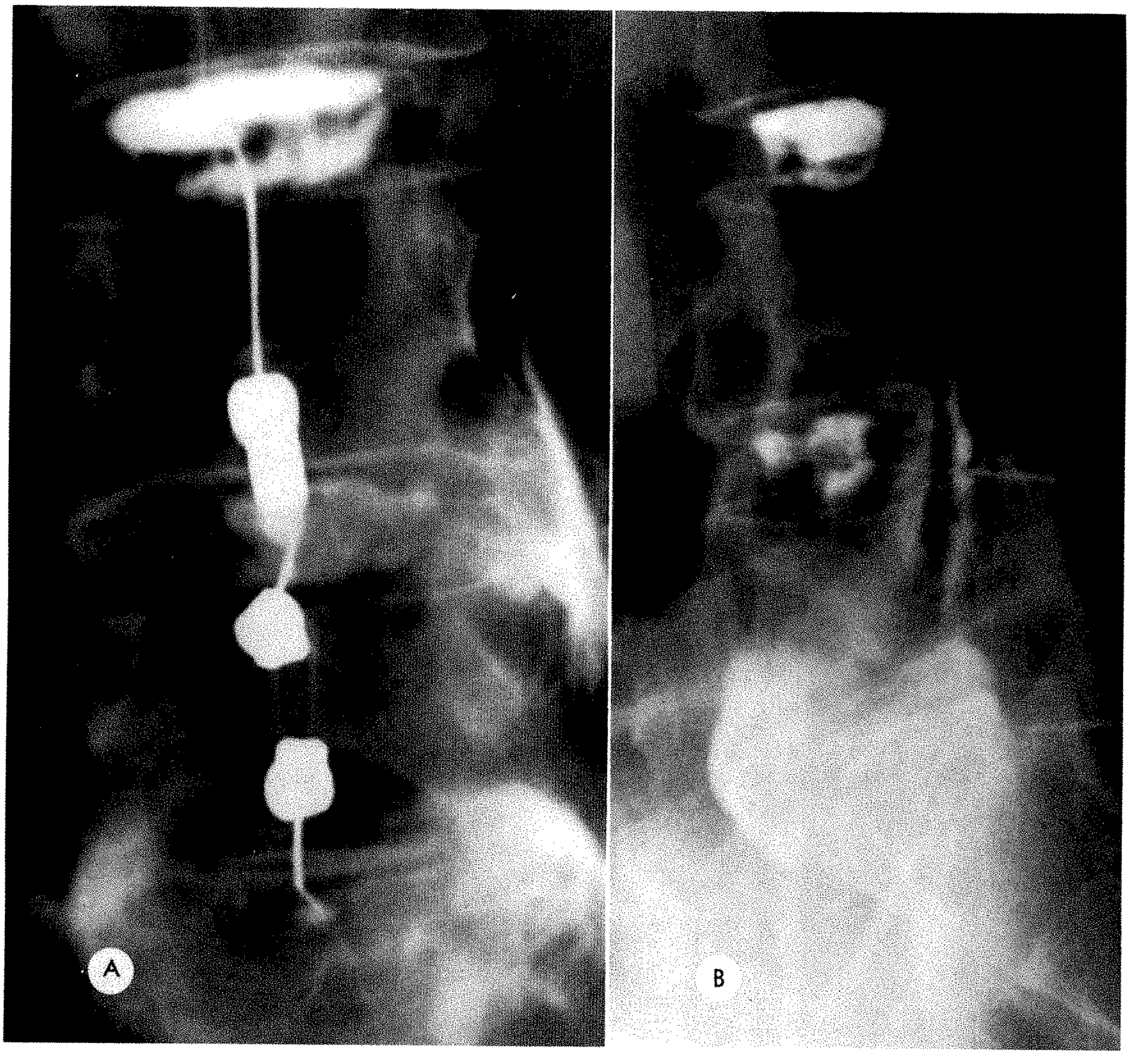

Fig. 5. (*A*) Anteroposterior spot roentgenogram appearing normal even though there is a slightly fissured and flattened nucleus at L3–4. Marked left lateral extension of nucleus substance with elevation of lateral longitudinal ligament in spite of central needle placement with 2 cc. of contrast material injected at L4–5. L5–S1 shows generalized nucleus degeneration with extension in all directions to the longitudinal ligaments. (*B*) Lateral view of same patient showing above findings in different plane.

ing a finding positive obviously will continue to feel that interpretation of diskography is difficult and unreliable. Combined myelography and diskography are used in our department for cases where the clinical findings are inconclusive.

Many do not accept the entity of diskogenic pain and accept only the classic physical compression of the nerve rootlets by a free fragment as a cause of clinical symptomatology. Lindblom,[11,12] Fernström,[6] and Walk[15] have elaborated on the etiology of diskogenic pain. It is far more common than simple free fragment etiology of pain and can be summarized as being a foreign body or chemical reaction resulting from the nucleus content reacting with the nerve rootlets.

As intimated above, diskography, in our department, is used primarily for evaluation of diskogenic pain. We use it in the free fragment group only for the purpose of

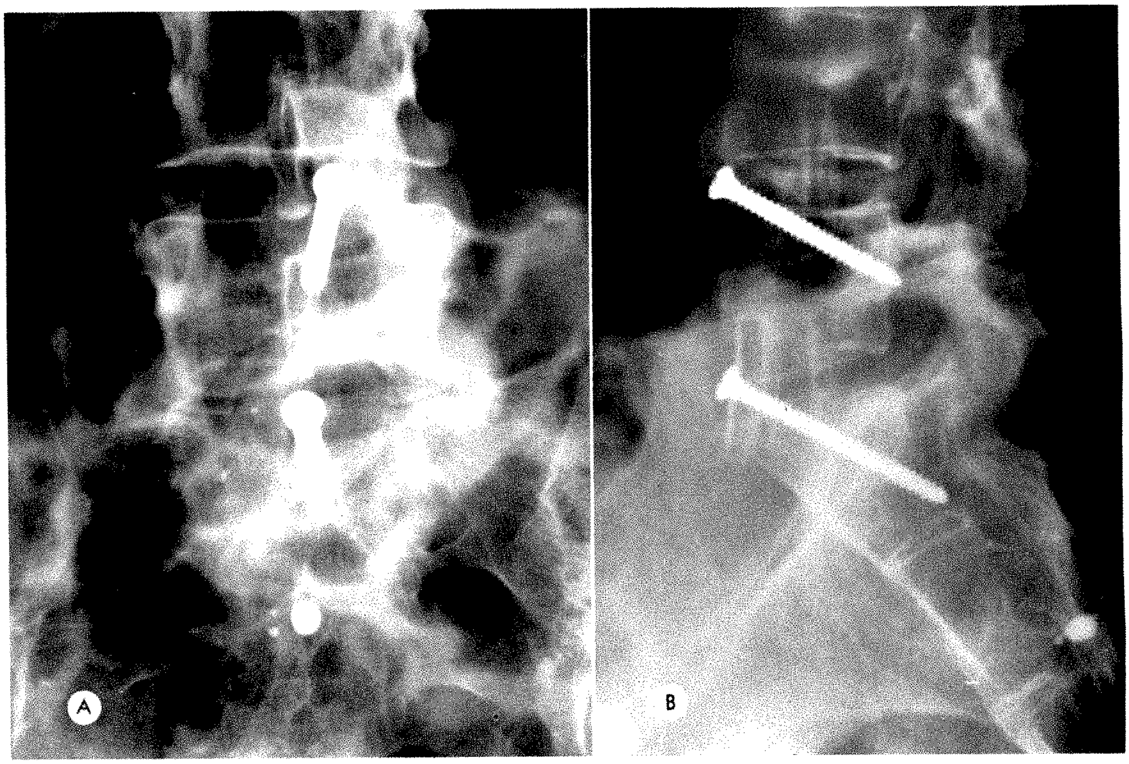

Fig. 6. (*A* and *B*) Same patient as in Figure 5, *A* and *B*, showing postoperative anterior spinal fusion bone plugs fixed by means of metallic screws at L4-5 and at L5-S1.

including or excluding the spaces above or below the obviously involved interspace. If 3 or 4 levels are pathologic, surgery at the classically involved space alone will not result in complete remission. With extensive involvement, one might advise against conventional posterior laminectomy because of the likelihood of poor results due to generalized disk disease.

With the advent of the anterior surgical approach to disk surgery,[8,9] diskography is finding even more application at the Mt. Sinai Hospital. Now cases with multiple levels of involvement are being re-evaluated for anterior spinal fusion. This applies not only to the posterior protrusion cases but also to those with extension of nucleus substance beyond the annulus confines bilaterally or anteriorly.

No attempt will be made to discuss the role of the anatomically contracted osseous spinal canal in the low back pain syndrome.[4] Likewise, the place of injection of hydrocortisone[5] or lytic agents[14] through disk needles will not be discussed.

SUMMARY

A more enthusiastic attitude in favor of diskography is advocated in view of the fact that low back pain is one of the most frequent complaints brought to the attention of physicians.

The technique used and modified from the original as described by Lindblom is reviewed.

The results of diskographic examinations of 2,320 patients between 1955 and 1964 are summarized in Tables I and II. Some 6,784 disks were injected.

Normal disk interpretation is emphasized. Three pathologic categories are described and illustrated.

Combined myelography and diskography are advised for the evaluation of the low back pain syndrome. Diskography should play a more important role with the advent

of the anterior surgical approach to disk disease.

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INTRAOSSEOUS VERTEBRAL VENOGRAPHY IN THE DIAGNOSIS OF LUMBAR DISK DISEASE*

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DEMONSTRATION of the vertebral venous plexus following the injection of contrast agent into the posterior spine of the vertebrae was reported by Fischgold *et al.*[3] in 1952. Since then, this procedure has been performed in the evaluation of pathologic processes by various investigators.[6,8,10,14,19] The only two large series evaluating the technique in the diagnosis of herniation of lumbar disks bear conflicting testimony. Although Schobinger[15] acknowledged that intraosseous vertebral venography yielded roentgenographic data somewhat more difficult to interpret than myelography, he and his collaborators[17] later reported correct localizations of disk protrusions in 30 of 33 patients by intraosseous venography. In contradistinction, Nathan and Blum[12] performed vertebral venography in 31 patients of whom 11 were lumbar disk suspects. They did not report positive localizations and concluded "that the variability of filling of the vertebral venous plexus makes the significance of non-filling of the plexus secondary to herniation of the nucleus pulposus questionable. Myelography is more certain and a more easily applicable technique for the demonstration of this pathology."

Myelography has been routinely utilized for the localization of lumbar disk protrusions for more than 25 years. The roentgenographic appearances of characteristic disk defects are widely appreciated.[1,4,9,13,18] Hanraets[5] has recently summarized opinions as to the accuracy of this technique.

Schobinger *et al.*[17] reported accurate myelographic localization of lumbar disk disease in 23 of 28 surgically verified cases, although they add that the technique was partially correct in 3 and suggestive in a fourth, and nonlocalizing in only a single instance. In a later paper Schobinger and Krueger[16] found venography and myelography to be equally accurate in all lumbar interspaces save the L 5–S 1 space, where venography was considered superior.

In an attempt to establish its true efficacy, intraosseous venography was performed on 40 successive patients with clinical histories and physical examinations suggestive of lumbar disk disease. This was followed by routine myelography. Two other patients having sacral tumors were similarly studied.

ANATOMY

An understanding of the vertebral venous anatomy is necessary for the correct interpretation of venographic studies. These veins in the human have been described in detail.[8,10,15,16,17] The roentgenographic appearance of these vessels in the dog has also been reported.[11,20] Crock,[2] analyzing micropaque roentgenographic studies on sections of canine vertebrae, demonstrated the intravertebral venous drainage. The largest venous channel in the posterior spine usually courses in the cephalad portion of the marrow space and bifurcates into larger channels in the cephalad portions of the laminae. These are joined by tributaries from the transverse processes. The enlarged venous channels pass into the pedicles where a vein usually courses out from the bone to join the intervertebral veins in the intervertebral foramen. Here, venous flow, subject to many regulating factors, may be either directed to the internal vertebral venous plexus in the epidural space (epidural veins or veins of Batson) or to the external vertebral plexus.

The lumbar epidural vessels usually

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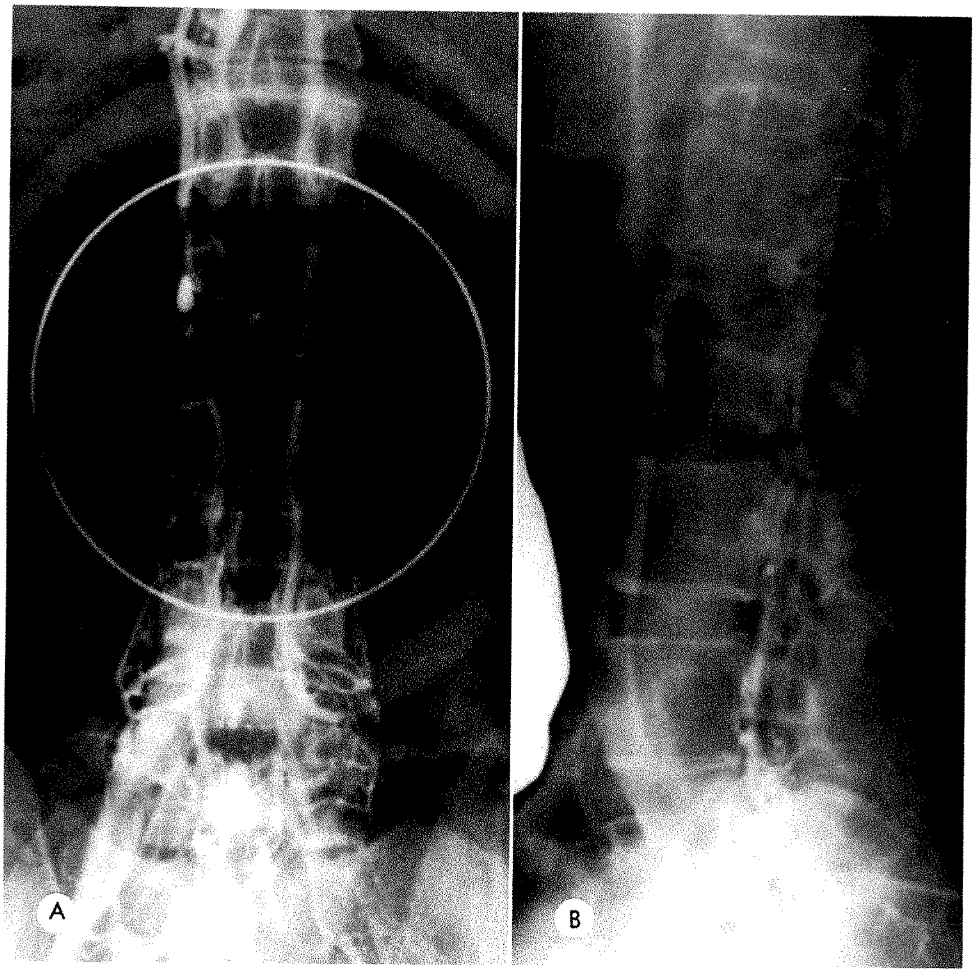

Fig. 1. (*A*) Anteroposterior roentgenogram shows good demonstration of internal and external vertebral vein in a normal person. (*B*) Normal lateral roentgenogram showing inferior vena cava, lumbar veins, internal vertebral veins and posterior external venous plexus.

course along nerve radicles and are connected to the adjacent segmental radicular veins by the intervertebral vein as shown in Figure 1, *A* and *B*. Caudal to the L 5 nerve root this radicular system is replaced by a series of small epidural veins coursing parallel to the neural tube and draining the sacral regions of the neural canal. In most cases the epidural veins cephalad to L 2 are large bilateral anterior vessels with transverse connections. This type of pattern is seen in lower mammals.[11,19]

MATERIAL AND METHOD

This series consists of 40 patients having syndromes clinically suggestive of lumbar intervertebral disk disease and 2 additional patients with malignancies, one with a sacral chordoma (Fig. 2, *A* and *B*) and the other with metastases of prostatic carcinoma to the sacrum (Fig. 3, *A* and *B*). These patients were seen over a period of 8 months at the Veterans Administration Hospital, Coral Gables, Florida, and represent a consecutive and not a selected series of lumbar disk suspects. The venographic technique used was as described by Schobinger *et al.*; only posteroanterior exposures were made. These authors and also Nathan and Blum were of the opinion that this view furnished more information than the lateral view. In the lateral view the ascending

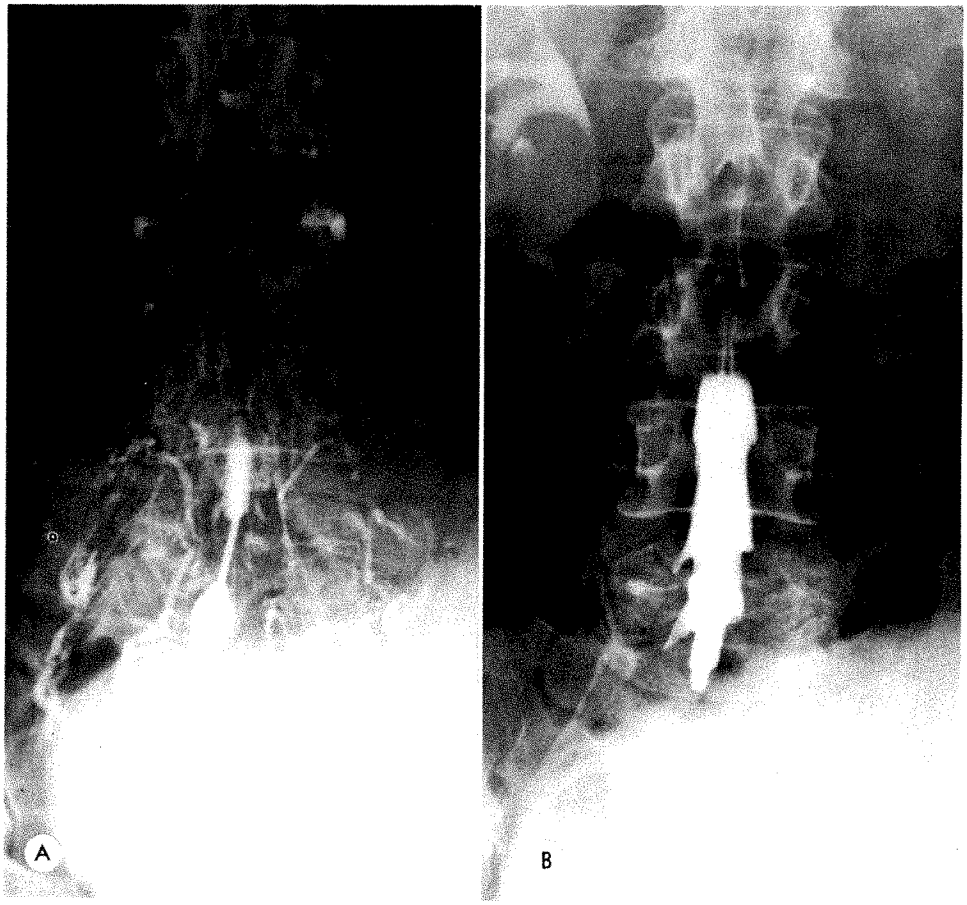

Fig. 2. (*A*) Intraosseous venogram shows attenuation and blockage of intervertebral vein at L 5 on left side. (*B*) Myelogram shows displacement of caudal sac to the right. The nerve root sleeve at L 5–S 1 is amputated. The sacral changes secondary to the chordoma are noted.

lumbar veins frequently project over the neural canal and usually only large lesions causing gross displacement of the epidural veins are demonstrable. Excessive extravasation of hypaque during the performance of the venography resulted in the myelography being postponed on several occasions; otherwise, the myelography was performed immediately following the venography. All myelograms were technically successful; two venograms were nondiagnostic because of inadequate filling and extravasation of contrast agent into the surrounding tissues. No ill effects were related to either of the procedures.

Decisions as to the time and interspace localization of the surgical exploration were based on clinical and myelographic criteria. The venograms were correlated with the operative findings and were interpreted by an independent observer without benefit of clinical, myelographic, or operative find-

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Fig. 4. (*A*) Intraosseous venogram shows complete block of intervertebral vein on right at level of L 4–L 5. There are dilated collateral veins demonstrated on right. The intervertebral vein at left is thinned. (*B*) Myelogram shows large extradural defect at level of L 4–L 5 on right. At operation, multiple extruded disk fragments were found.

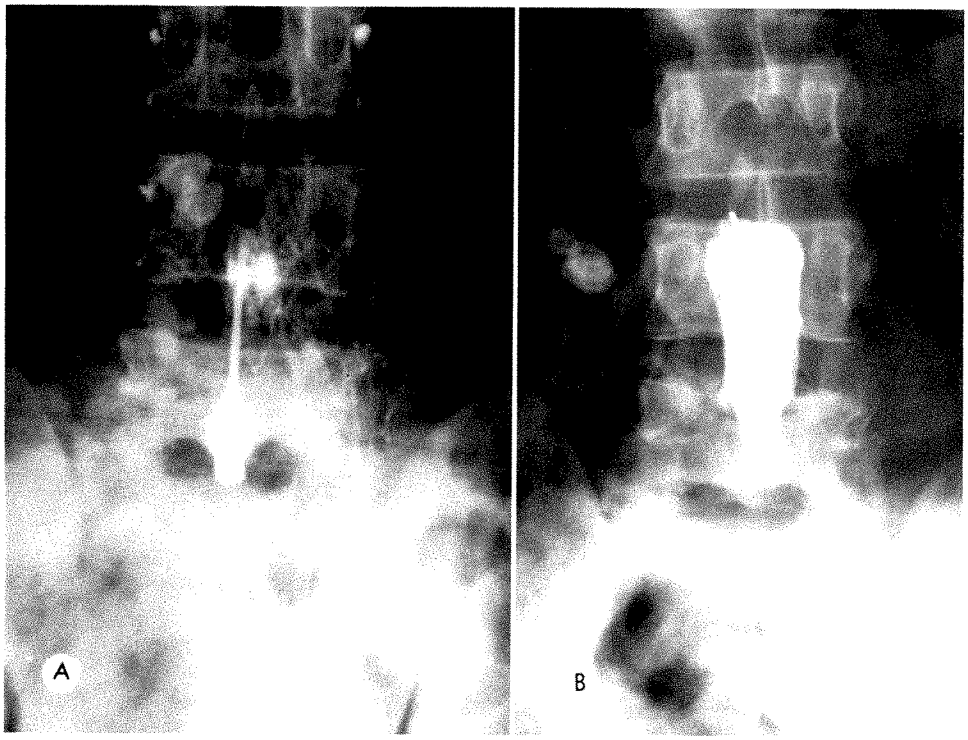
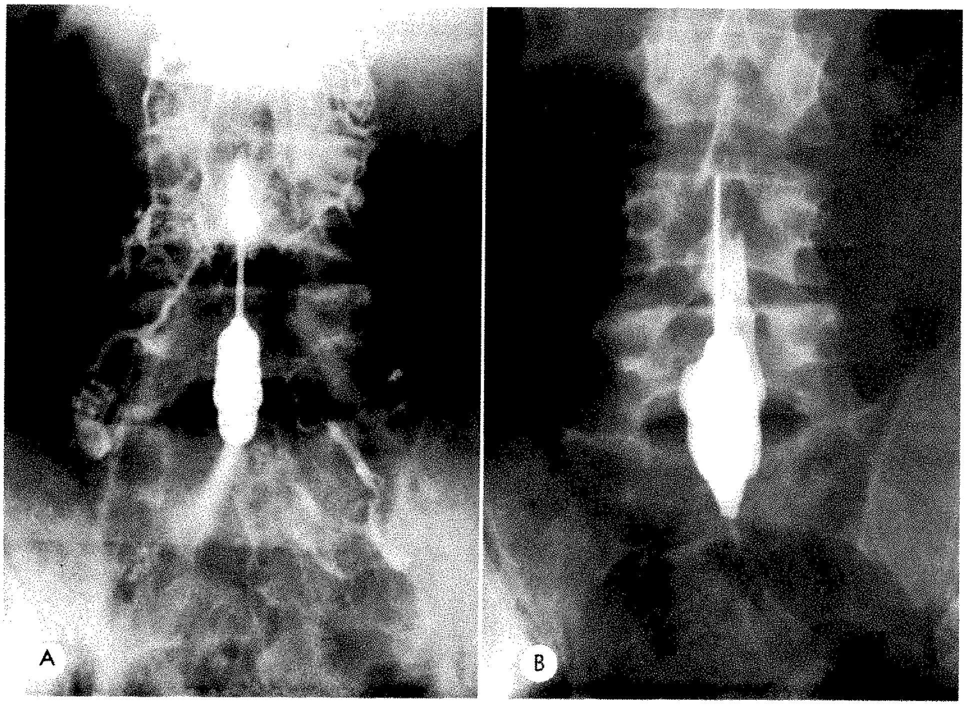


FIG. 3. (*A*) Intraosseous venogram shows complete block of epidural views at level of L 4–L 5. Block extends higher on right than left side. (*B*) Myelogram shows complete block at level of L 4–L 5. Extradural tumor extends laterally on right side almost to midportion of L 4. Characteristic osteoblastic changes of carcinoma of prostate noted in sacrum, ilia and L 4–L 5.



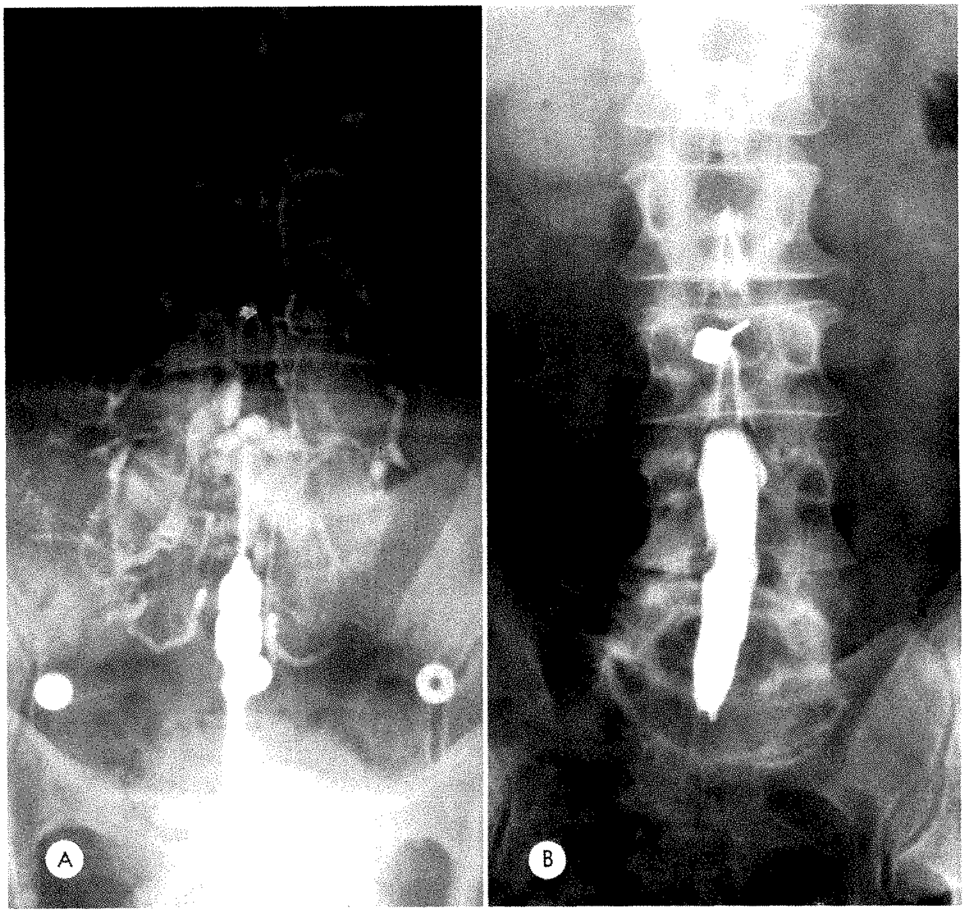


FIG. 5. (*A*) Intraosseous venogram shows block at L 4–L 5 on right side. There is phlebectasia of intervertebral below block and poor filling of intervertebral vein on right above block. (*B*) Myelogram shows extradural defect at L 4–L 5 on right side.

ings. Of the 40 myelograms made on herniated disk suspects, 29 showed disk protrusions, and 11 were read as normal. Twenty-six were surgically explored and the presence of a herniated disk was verified at the roentgenographically suggested level. Three individuals with positive myelograms were discharged when their pain disappeared with conservative therapy.

The important factors in venographic interpretation were: (1) localized failure to fill the internal vertebral (epidural) venous plexus, or narrowed caliber or absence of these vessels as they cross an interspace (Fig. 4, *A* and *B; 5, A* and *B;* and 6, *A* and *B*); (2) failure to visualize an intervertebral (radicular) vein as it courses through the intervertebral foramen (Fig. 6, *A* and *B*); (3) the presence of an abnormal curvature, irregular localized dilatation, or phlebectasia of the epidural veins (Fig. 7, *A* and *B*); (4) excessive caudal flow of the contrast agent. In such cases the external vertebral veins and the ascending lumbar veins are outlined several segments below the lesion as compared to solely one or two levels in the normal case. This excessive caudal venous demonstration is usually more prominent on the side of the disease process (Fig. 8, *A* and *B*); and (5) inability to fill the internal vertebral venous plexus when the injection is into the spine of the vertebrae immediately cephalad to the diseased interspace. When this happens, it is thought that the intervertebral vein is occluded at the median aspect of the inter-

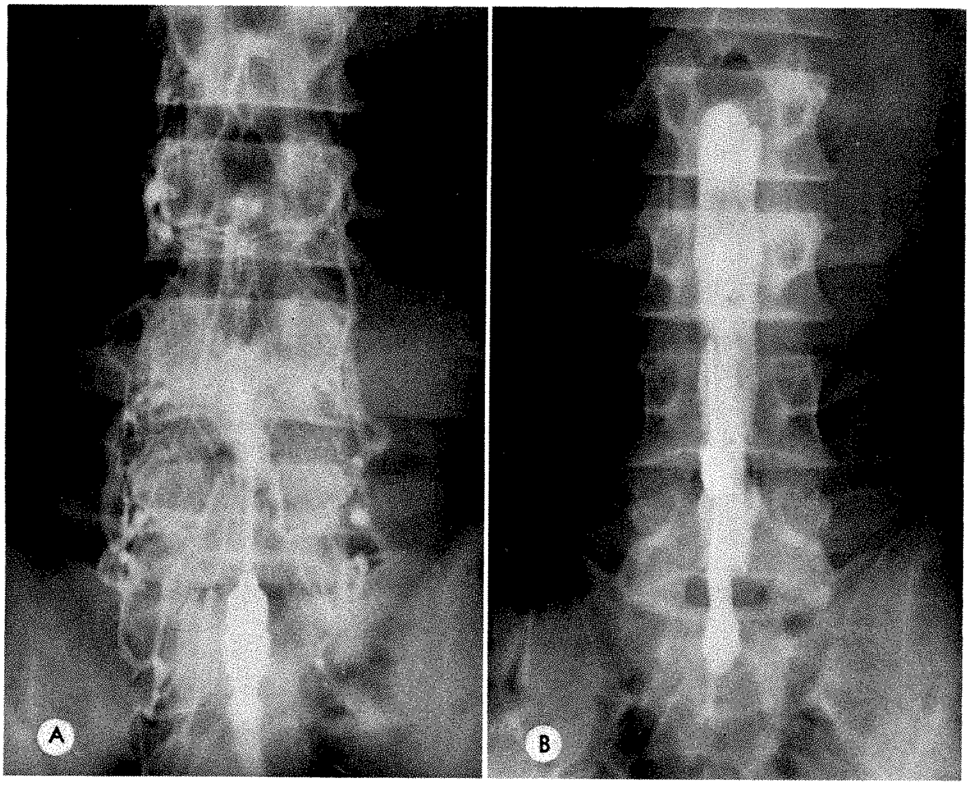


Fig. 6. (*A*) Intraosseous venogram shows excessive unilateral flow of contrast material on right. There is a lack of filling of intervertebral vein on left at L 5–S 1. (*B*) Myelogram shows herniated disk at L 5–S 1 on left side.

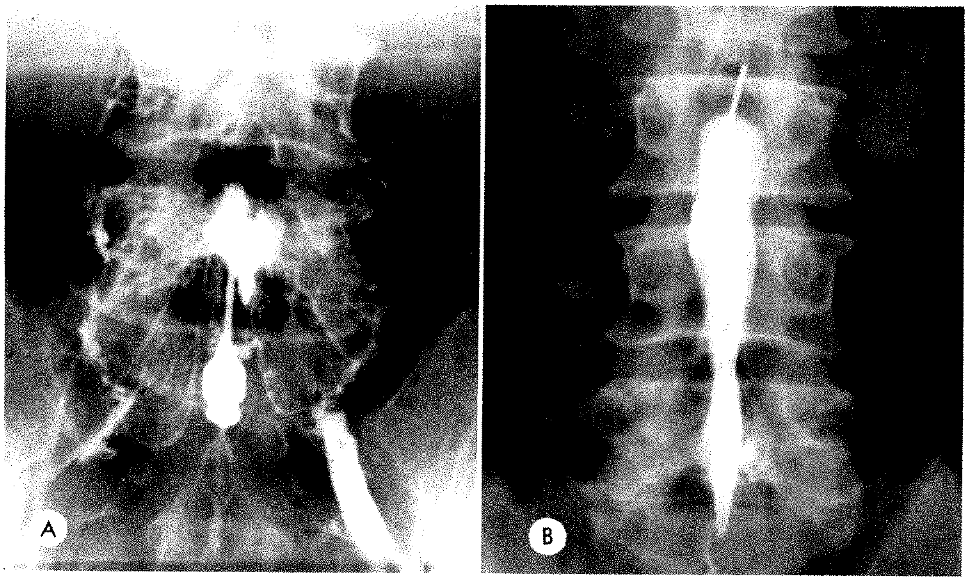


Fig. 7. (*A*) Intraosseous venogram shows block at L 4–L 5 with failure of filling of radicular vein bilaterally at this level. The intervertebral vein is thinned on the right but obliterated on the left. There is collateral circulation retrograde to circumvent the block. (*B*) Myelogram shows bilateral defect at level of L 4–L 5 with failure of filling of both nerve root sleeves at this level.

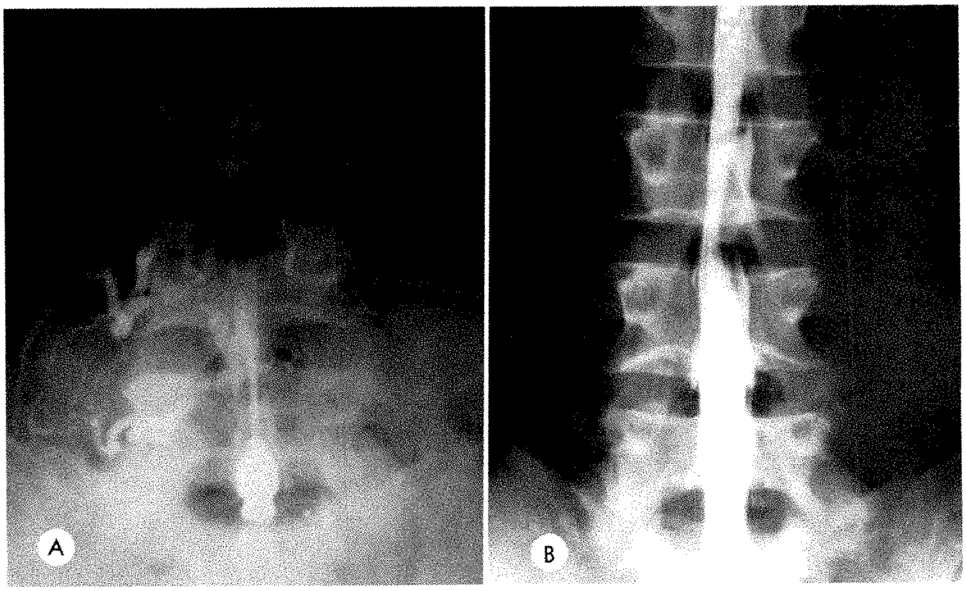


Fig. 8. (*A*) Intraosseous venogram shows phlebectasia of intervertebral and extravertebral veins. There is a block at L 3–L 4. There is less filling of veins on left side. (*B*) Myelogram shows extradural defect at L 3–L 4 on left side. Medial displacement and enlarged veins are well demonstrated.

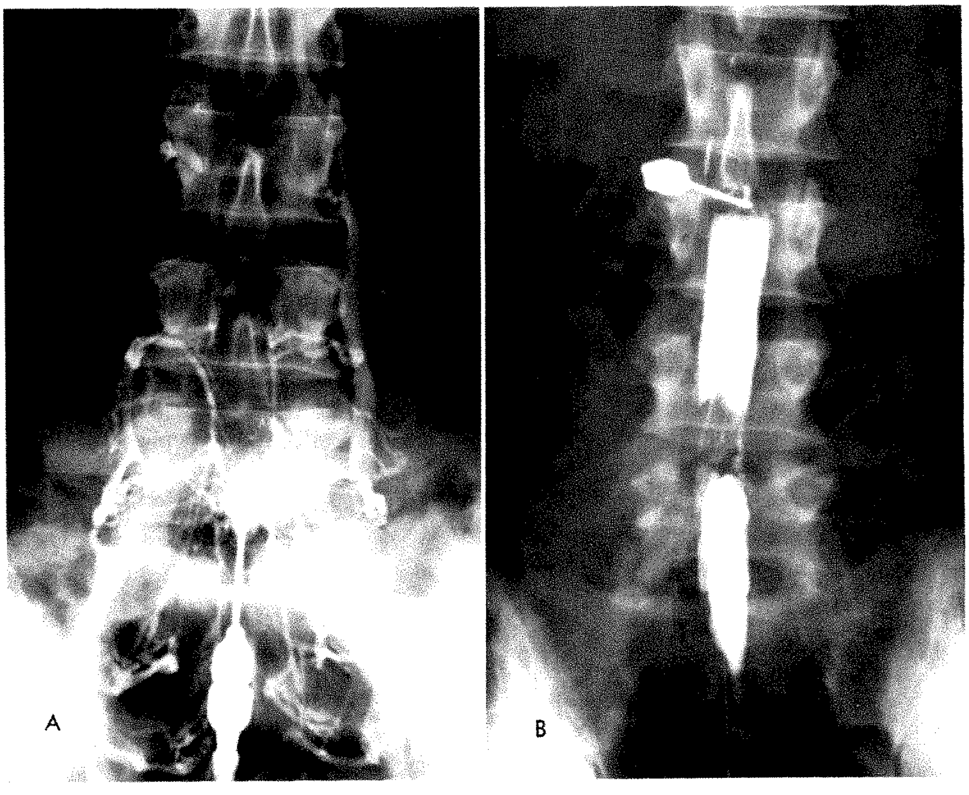


Fig. 9. (*A*) Intraosseous venogram demonstrates excessive caudal flow of contrast agent. There is abnormal curvature of intervertebral vein on right at level of L 4–L 5. (*B*) Myelogram shows large herniated disk at level of L 4–L 5 on right side.

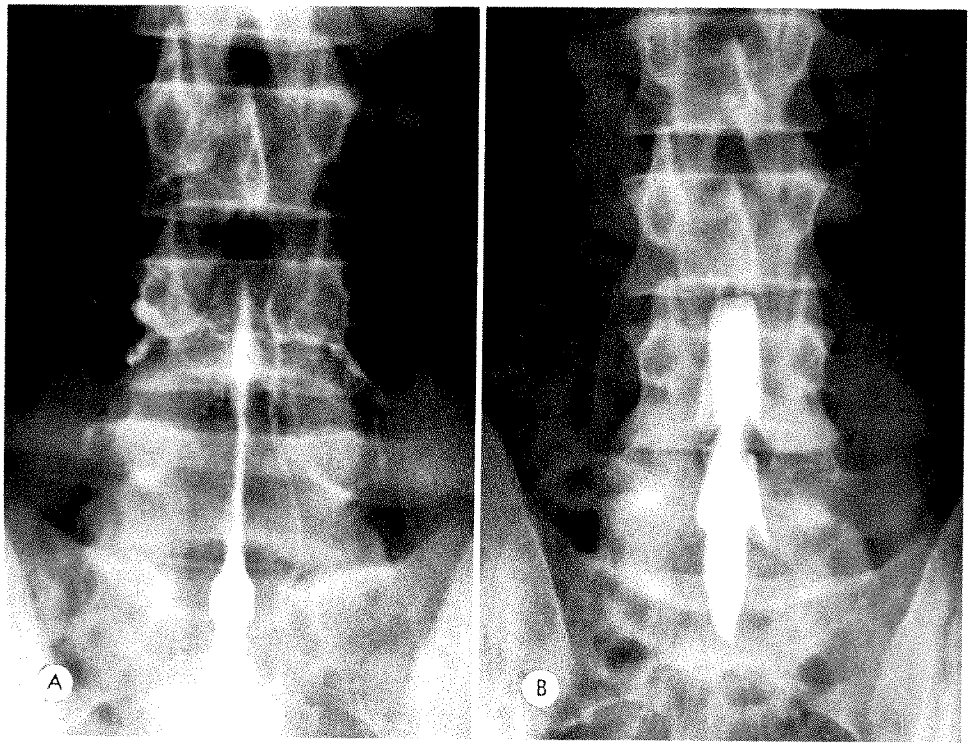


Fig. 10. (*A*) Intraosseous venogram shows block at L 4–L 5. Intervertebral vein is filled on left. Radicular veins are not filled on either side at L 4–L 5. (*B*) Myelogram shows bilateral defects at L 4–L 5; slightly more marked on left side. Midline disk found at operation.

vertebral foramen and all flow is directed into the external vertebral plexus (Fig. 10, *A* and *B*).

Vertebral venography is dynamic and the distribution of the contrast agent must always be considered on physiologic principles. Only after a review of the many vascular patterns obtained can some of the more subtle variations of the technique be appreciated.

The results of an impartial review of the venograms are presented in Table I. Thus, it is seen that intraosseous vertebral venography can delineate a lumbar disk herniation; however, not with the certainty attributed to myelography. The interruption of venous drainage by the scar resulting from previous laminectomies (prior to admission) were considered a factor in the interpretation of 2 venographic failures, 1 false positive and 1 false negative (Fig. 11, *A* and *B*). Another false localization can be attributed to developmental hypoplasia of the spines of L 4 and L 5 (Bantu spine), necessitating injection into the L 3 spine with resultant nonvisualization of the venous pattern at the pathologic L 5 level.

On 2 occasions the venogram was very suspicious not only at the pathologic level but also at other levels. In such instances, the myelographic abnormality should dictate the level of surgical intervention. Venograms showing a complete pattern of the intervertebral vessels and the epidural vessels from L 2 to S 1 level have invariably

Table I

Venographic Interpretation	Correct	Incorrect
Herniated Disk at Specific Interspace	23*	11*
No Herniated Disk	4	4

* Includes 2 cases with localization at both correct and incorrect interspaces.

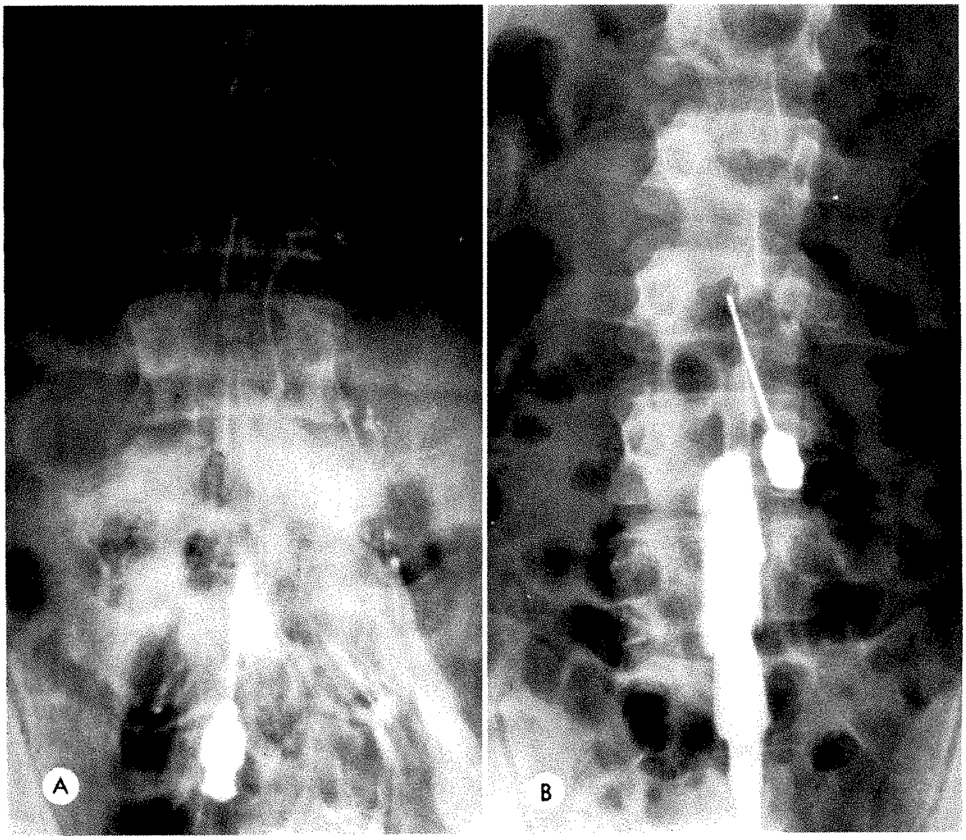


FIG. 11. (*A*) Intraosseous venogram is difficult to evaluate as the patient had previous interlaminar explorations at L 4–L 5 and L 5–S 1 on the right. However, the intervertebral vein is well demonstrated on left at L 5–S 1. Intervertebral vein is not filled on right at L 5–S 1 and the epidural vein on the left is displaced laterally. From this study it was felt that there was a recurrent herniation at L 5–S 1. (*B*) Myelogram demonstrated extradural defect at L 5–S 1 on left with elevation of nerve root sleeve. At operation, a bulging disk with an intact annulus was found.

been associated with a normal myelogram. In this respect, lumbar intraosseous venography as a screening test for disk subjects may be justified. However, if the history, clinical findings, and failure to respond to conservative treatment suggest disk disease, myelographic studies should be made.

All too often, the venous pattern is poorly differentiated, especially in the aged and in those with osteoporosis (Fig. 12, *A* and *B*). This may represent an aspect of aging. Hulth[7] observed that in osteoarthritic hips there is retarded venous drainage via the femoral shaft, whereas in the normal hip venous drainage is rapid and directly into the circumflex veins of the hip.

SUMMARY

A consecutive series of 40 patients suspected of having lumbar disk herniations and 2 patients with massive sacral tumors were investigated by myelography and intraosseous vertebral venography. Both techniques were used without untoward reactions.

All 26 patients undergoing operative removal of the herniated or ruptured disk had prior localizing myelograms. In 20 of these cases, significant alterations of the usual venographic pattern were present at the site of the abnormal disk. In both tumor cases, the epidural vessels filled only to the level of cephalad tumor extension. In this series of venographic studies, pathologic

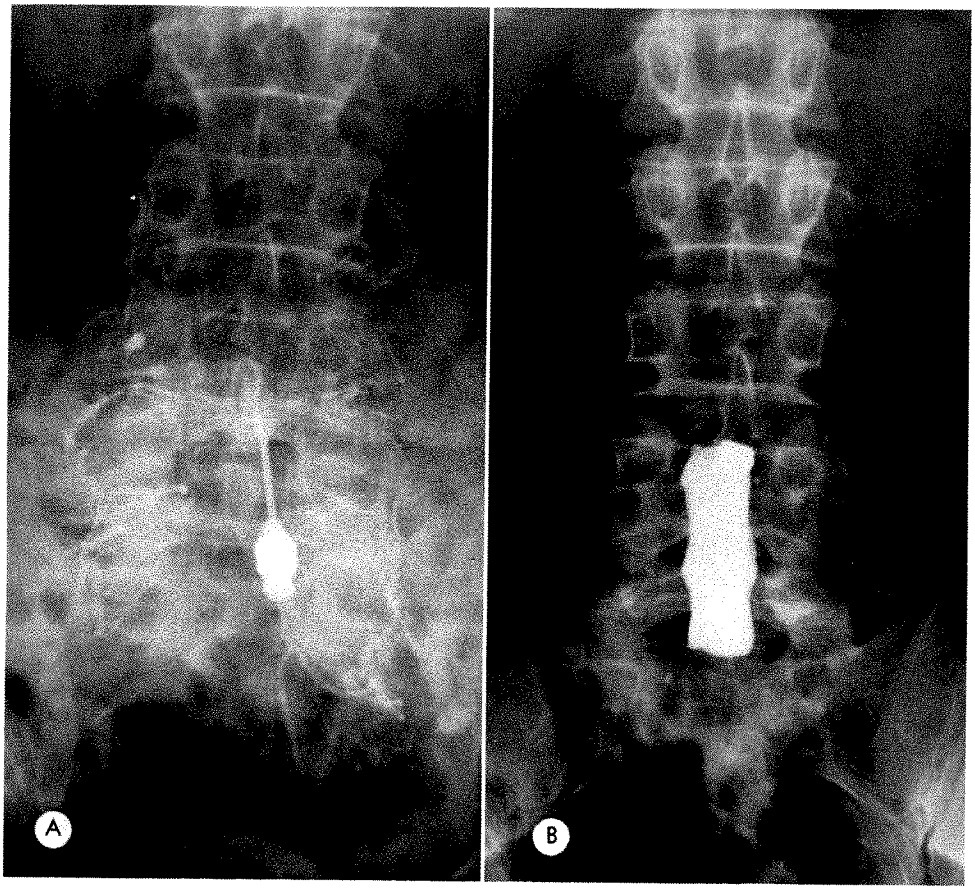


Fig. 12. (*A*) Intraosseous venogram shows predominantly extravertebral filling. Intervertebral vein is quite small at all levels. Radicular veins are not well demonstrated. (*B*) Myelogram shows a wide canal. No evidence of extradural defects.

changes were suspected at 14 additional interspaces where clinical, myelographic, and surgical evaluation failed to establish the presence of disease. However, in 3 of these cases, clinical improvement resulted in discharge from the hospital prior to surgical confirmation of probable disk herniations.

Although venography is a useful diagnostic aid, this study suggests that the presence and site of disk disease can best be localized with myelography.

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SPINAL LIPOMA*

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SPINAL lipomas are rare, and constitute only 1 per cent of all the primary intrathecal neoplasms.[5] To date, less than 60 cases have been reported in the English medical literature with reference to only one in the radiologic literature.[8]

In all but a few cases, the diagnosis was not made prior to surgical intervention,[2,4,14] although clinical and roentgenographic findings do exist to make the preoperative diagnosis possible in a high percentage of cases.

A review of records at our institution disclosed 2 cases of spinal lipoma in adults and 12 in children. The children all presented with posterior masses in the lumbar region which were classified as meningiolipomas. They had fatty tissue within the herniation that extended into the spinal canal. These cases are similar to those reported by Bassett[1] and will not be included in this article.

REPORT OF CASES

Case i. This 35 year old white male presented to our Institution with an 8 year history of loss of strength throughout the right leg and loss of sensation about the inner aspect of the right knee. On admission to the hospital, the weakness had developed to such a point that he frequently stumbled when walking and found it necessary to drag his right foot. Because of the progressive nature of the weakness, he had sought medical aid in the past and atrophy of the musculature of the right leg had been documented. He had no back pain at any time. He did note that he could not completely empty his bladder, but he had not had any bowel symptoms. Because of a past history of trauma to the right buttocks requiring surgical drainage of a large hematoma, the impression on admission was that the patient had traumatic degeneration of the right sciatic nerve.

Physical Examination. Examination of the patient's back revealed a full range of motion with no pain or tenderness. There was obvious diffuse atrophy of the right leg with marked weakness of the right quadriceps and the extensors of the right foot and toes. Fasciculations of the muscles of the right leg were present.

Spinal puncture was within normal limits. Standard lumbar roentgenograms were obtained and revealed evidence of early erosion of the right pedicles of L1 and L2. Total myelography demonstrated an intramedullary lesion of the upper lumbar spinal canal at the L1–L2 level (Fig. 1 and 2).

Operative Findings. A lipoma of the cauda equina was found which was anterior to the cord, particularly on the right side. It was at the distal aspect of the conus medullaris and was intermingled with the cord (Fig. 3). Surgical removal was not possible and only a small piece was removed for biopsy. Pathologic examination showed lipoma.

Postoperative Course. Immediately after the operation, the patient was able to move his right foot and toes. Control of urinary stream returned after 2 days. He was started on immediate ambulation and was noted to have diffuse weakness of the right leg as well as loss of sensation with a depressed right patella and achilles reflex. This was thought to be worse than it had been preoperatively. Two months following surgery, his general condition was improved although he did have a marked limp. The right patella and achilles reflexes remained absent and there was hypesthesia in the right L3–L4 area. Follow-up after another 2 months' interval showed no change in his condition.

Case ii. A 30 year old white male had noted progressive weakness of the left leg for a period of 4 years. The left knee would buckle after prolonged standing. One year prior to admission, the left calf was noted to be considerably smaller than the right. On admission to this Institution, he complained of paresthesia along

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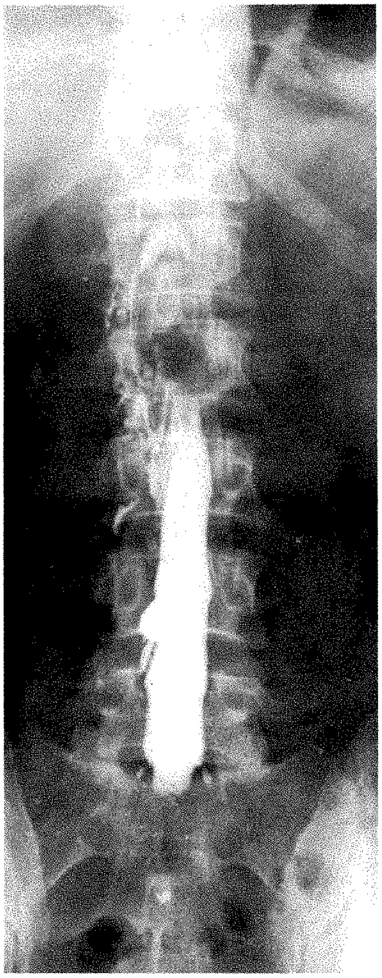

FIG. 1. Case 1. Upright myelogram. Note length of lesion and minimal erosion of pedicles of L1 and L2.

the lateral aspect of the left ankle and foot, especially in the 4th and 5th toes, and weakness of the left foot to the extent that he could not stand on the toes of his left foot. He denied bladder, bowel and sexual difficulties.

Physical Examination. There was weakness and atrophy of the left leg. Strength and leg-raising maneuvers were not painful. There was normal range of motion of the back with no associated tenderness. The deep tendon reflexes were absent on the left. Para-anal hypesthesia was present.

Spinal puncture revealed a protein of 93 mg. per cent with normal pressure. No abnormal blood cells were seen. Roentgenograms of the sacrum revealed a large left sacral defect associated with a wide sacral canal (Fig. 4). Myelography was carried out and revealed a congenitally long spinal cord and outlined the periphery of the tumor (Fig. 5).

Operative Findings. When the roof of the sacrum was removed, it was seen that the spinal cord descended to the third sacral segment and there, a bulbous lipoma was encountered. The nerve roots were seen to go upward and exit through the appropriate foramina (Fig. 6). Surgical excision was impossible without sacrificing the sacral nerve roots and biopsy only was performed.

Postoperative Course. Immediately after the operation, the patient had some radicular pain. Complete bladder control returned in approximately 10 days. Follow-up at 1 and 2 month

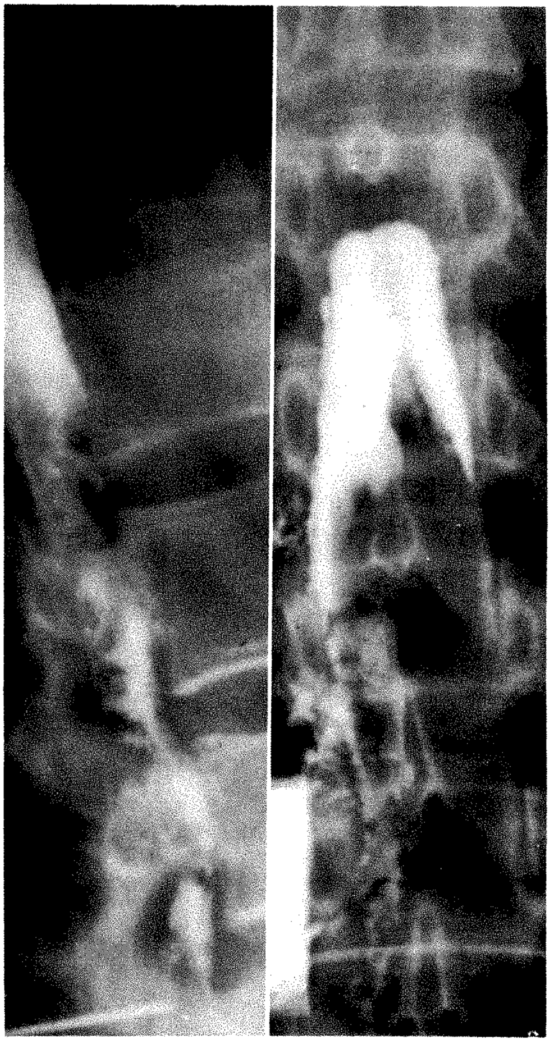

FIG. 2. Case 1. Superior margin of intrathecal lesion is well outlined.

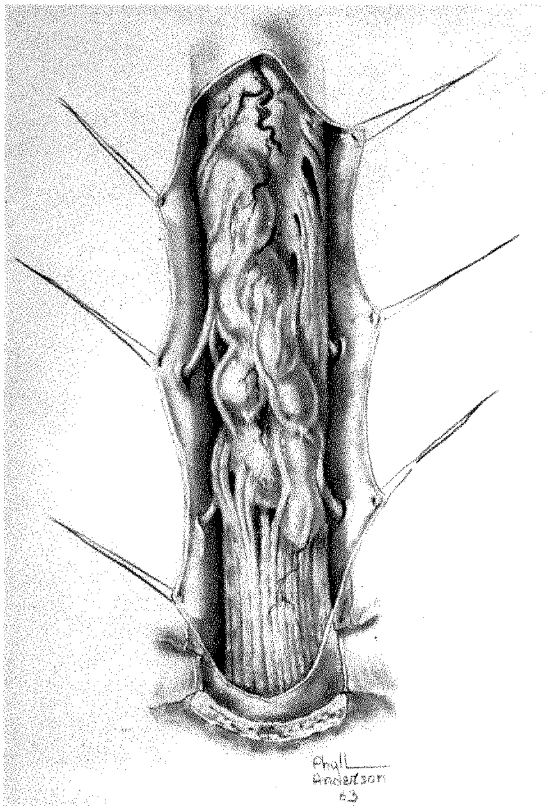

FIG. 3. Case I. Drawing showing the appearance of the lesion at surgery. Note the length of the lesion and the displacement and stretching of the nerve roots.

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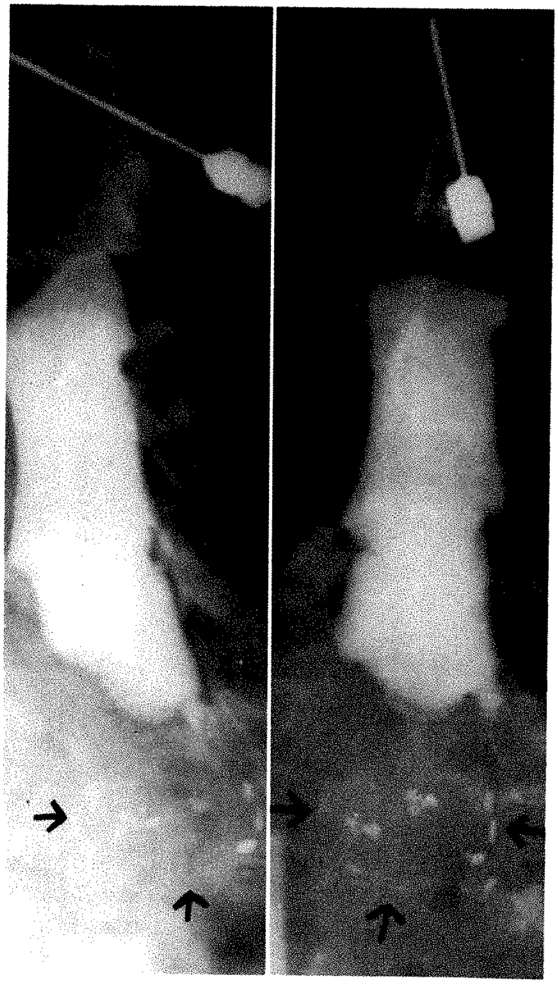

FIG. 5. Case II. Droplets of contrast material outline the lesion (arrows). An associated congenital anomaly was the presence of a spinal cord extending down to the third sacral segment.

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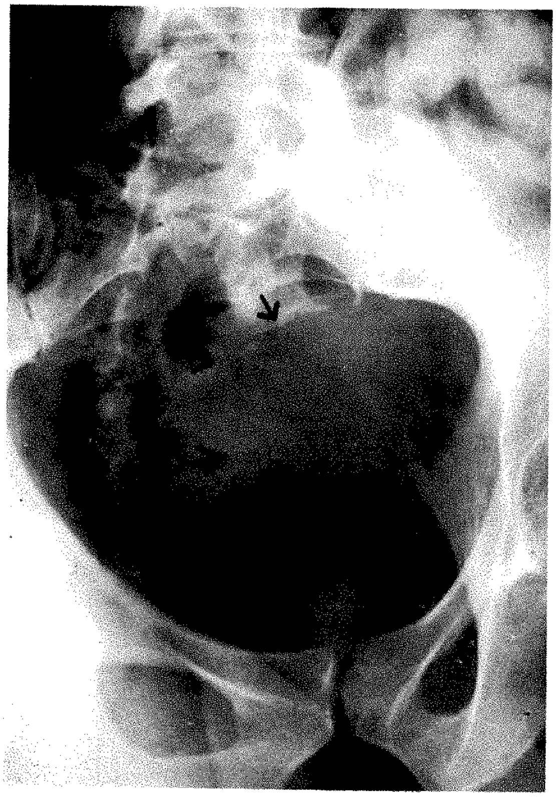

FIG. 4. Case II. Plain roentgenogram of the sacrum showing the large bone defect (arrows).

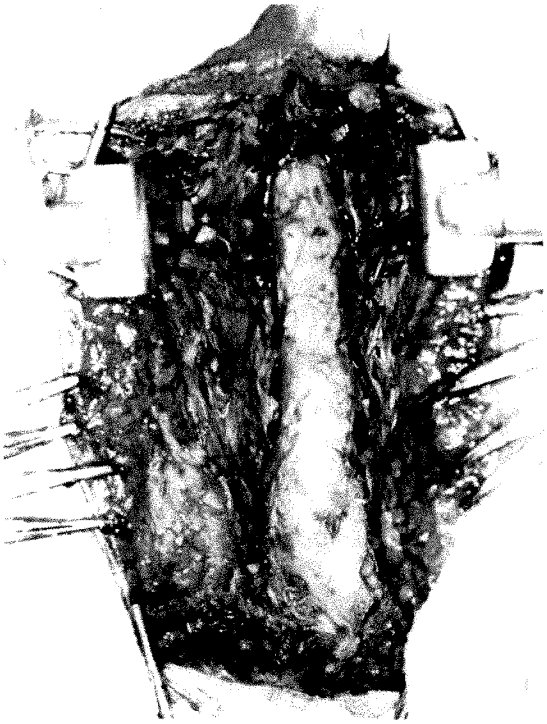

FIG. 6. Case II. Photograph showing operative findings. Note the nerve roots coursing upwards.

intervals postsurgery showed essentially no change in his clinical status.

DISCUSSION

CLINICAL FEATURES

Numerous theories have been advocated as to the origin of these tumors.[11] They are thought to be mesenchymal,[5] rather than neural in origin. Microscopically, they are composed of adult adipose cells identical to fatty tumors elsewhere in the body.

A congenital origin has also been suggested because of their frequent association with various congenital anomalies. This is demonstrated in our Case II where the spinal cord was found to extend to the third sacral segment and a large defect in the sacrum was present. Of all the congenital anomalies reported in association with spinal lipomas, the most common is subcutaneous lipoma.[2]

These tumors have been reported equally in both sexes. The age distribution is widespread although predominantly they are found in the young. The lesion has most often been reported in the lower cervical[11,12,16] and thoracic[3,7,9,10,13,15] areas. The tumor is usually attached to the pia and although its bulk may be extramedullary, it appears to have an intramedullar component, and delineation from neural tissue is not grossly apparent.

These tumors produce pressure symptoms, but due to their soft nature, they can attain considerable size which is out of proportion to the relatively moderate neurologic deficit. Several cases have been reported in which the tumor extended over the entire spinal canal.[12]

The neurologic deficit may be vague and of long duration. The average length of symptoms is 4 years with some patients having symptoms for as long as 8 to 12 years. One of the striking clinical findings is the lack of associated nerve root pain. In the thoracic and lumbar tumors, a spastic paraparesis eventually develops. If the cervical region is involved, flaccid weakness of the arms may be an associated feature. The spinal protein may be normal or elevated, but the pressure is not elevated, as a rule, regardless of the size of the tumor. One report states that a complication of spinal puncture in these cases is the aggravation of the neurologic symptoms.[2]

ROENTGENOGRAPHIC FINDINGS

Plain roentgenograms of the spine may show erosion of the pedicles and an increase in the interpedicular distance. On myelographic examination, there may be a very long intrathecal mass with almost total block of the flow of the contrast material. The combination of the large cord mass with an almost total block of the contrast substance, in the absence of neurologic deficit of comparative severity, appears to be the characteristic pattern of these lesions. The differential diagnosis would include syringomyelia, slow growing glioma, subarachnoid cyst, and the degenerative diseases of the spinal cord.[6] Careful correlation of the clinical features and contrast studies will frequently be impressively accurate.

OPERATIVE FINDINGS

At operation, an intrathecal lipoma of rather large dimension is frequently encountered which may extend many segments and is encapsulated by thick fibrous tissue. Extension into the spinal cord is usually present. The tumors should be totally excised when possible, but often this cannot be accomplished and decompression constitutes the only surgical therapy. Decompression is almost always of symptomatic benefit. The primary function of operation is to halt the progression of neurologic deficit.

SUMMARY

The clinical picture of a slowly developing painless spastic paraparesis with roentgenographic evidence of an extensive intrathecal spinal mass is thought to be characteristic of an intradural lipoma. The 2 cases which are reported here are thought to demonstrate the long duration of a neurologic deficit and the characteristic absence of pain. Although these tumors can seldom be totally excised, early diagnosis with surgical intervention is almost always of benefit in stopping the immediate progression of neurologic symptoms.

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MULTIPLE LESIONS OF THE SPINAL CORD*

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LITTLE attention has been given in the medical literature to cases of multiple lesions of the spinal cord.[1,3] In order to assess the incidence, nature and diagnostic problems connected with these lesions, we have reviewed a series of 952 patients operated on at the Istituto Neurologico di Milano.

The series was divided into 3 groups according to the type of lesion:

Disk prolapses	583 patients
Tumors	312 patients
Miscellaneous	57 patients

DISK PROLAPSES

The 583 patients of this group had 669 disk prolapses with the following distribution:

Cervical disk prolapse	30 cases (4.50 per cent)
Thoracic disk prolapse	13 cases (1.95 per cent)
Lumbar disk prolapse	626 cases (93.55 per cent)

In 82 patients (14 per cent) there were multiple disk prolapses. One patient had 2 cervical disk prolapses and another had 1 cervical and 1 thoracic disk prolapse. In the remaining 80, the lumbar disks were involved, 76 patients having 2 disk prolapses, 3 patients 3 disk prolapses, and 1 patient 4 disk prolapses. The most frequent associations were: L4 and L5 in 55 cases (69 per cent), L3 and L4 in 14 cases (17 per cent), and other sites in 11 cases (14 per cent).

The multiplicity of the disk prolapses is the most frequent cause of operative failures. Thirty patients, operated on elsewhere for lumbar disk prolapse, without myelographic studies having been performed, were admitted to our hospital because of continuance or aggravation of the disturbances. In 9 of these (30 per cent), myelography and operation disclosed another disk prolapse at a site other than the one operated upon. One should bear in mind that one obstructive disk prolapse may mask another disk prolapse. This was observed in one-third of 38 cases in our series. Also, a verified ruptured disk at L4–5 with an associated anomaly at the lumbosacral level is suggestive of abnormality in more than one disk.[2]

Of significance is the fact that the lowest percentages of multiple disk prolapses reported in the literature are in groups of patients who were operated on following little or no utilization of myelographic studies, while the highest percentages are in series studied extensively by this means.

SPINAL CORD TUMORS

In the 312 patients who were operated on for spinal cord tumors, the distribution as to type was as follows:

Neurofibromas	87 patients
Meningiomas	82 patients
Gliomas	70 patients
Metastases	25 patients
Congenital tumors	17 patients
Lymphomas	15 patients
Neurofibromatosis	7 patients
Miscellaneous	9 patients

Multiple localizations were observed in 14 patients (4.50 per cent); in 12, these represented tumors, and in 2, tumors in combination with other lesions. The tumor group comprised: 7 cases of more or less disseminated spinal neurofibromatosis, chiefly in the cervical and lumbar regions; 2 cases of neurofibromas with other nodules attached to the root of the main tumor; 2 cases of meningiomas, each with 2 thoracic tumors; and 1 case of 2 ependymomas (both removed), one in the cervical and one in the midthoracic region. In the combined group were: 1 case of meningioma overlying a thoracic disk prolapse; and 1 case of thoracic neurofibroma associated with a spinal cord angioma, 2 metameres up.

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MISCELLANEOUS

In the group of 57 patients with miscellaneous lesions, 17 patients had epidural granulomas, 5 had extradural cysts, 1 had choroido-ependymal cysts and 34 patients had spinal cord angiomas.

Multiple lesions were found in 4 cases (7 per cent). One patient had an epidural granuloma in both the cervical and thoracic site; 1 had a double extradural cyst of the thoracic region; 1 had multiple choroido-ependymal cysts; and 1 patient had a thoracic angioma associated with lumbar disk prolapse.

This group does not include the cases of arachnoiditis observed around the disk prolapses, tumors and other spinal cord diseases, since these are regarded as complications of the principal lesion.

DISCUSSION

Of 952 patients who were operated on for spinal cord diseases 100 (10.50 per cent) had multiple lesions. The distribution according to type of lesion was as follows:

Disk prolapses	82 patients
Tumors	14 patients
Miscellaneous	4 patients

As may be seen, disk prolapses formed the great majority with 82 per cent, spinal cord tumors and other conditions accounting for the remaining 18 per cent.

With the exception of neurofibromatosis, which by definition implies multiplicity, these lesions present difficult diagnostic and practical problems.

Clinically, they may occur adjacent to each other, as is the case with disk prolapses, or they may be many metameres apart. The diagnosis is further complicated by the variations in the site and size of the lesion. In the spinal cord these difficulties may be insurmountable, for generally the symptoms due to the larger lesion tend to supersede those of the lesser lesion, and in the case of two lesions of equal size the uppermost lesion tends to produce the more pronounced symptoms. In the cauda equina the situation may be more favorable, especially as regards prolapses, since the manifold character of the roots affected will permit recognition of compression in several sites even if the exact location of the lesion cannot be determined.

From the neurosurgical point of view, failure to accurately diagnose these lesions may lead to unsuccessful operation or to partial and temporary results. In the more advanced or widespread lesions surgery may be directly contraindicated.

Roentgenologically, demonstration of multiple lesions of the spinal column is definitely possible. In the present series positive findings were noted in 4 cases of neurofibromatosis, in 1 case of double extradural cyst and in 1 case of multiple choroido-ependymal cysts. In cases of disk prolapse roentgenologic examination was found to be more useful in excluding other vertebral changes than in indicating the presence of prolapse.

Systematic myelography is the procedure of choice for demonstrating these lesions. The method is also useful in clarifying some of the atypical neurologic symptoms as, for example, in lesions of the upper spinal cord, which can give rise to sharp or burning pain radiating to a lower extremity, similar to the pain produced by a lumbar disk prolapse or other diseases of the cauda equina. Scott[5] noted such atypical symptomatology in 6 cases, or 7 per cent, of the 76 cervical and thoracic spinal cord tumors operated on at his hospital. We have likewise observed cases of this kind, although our percentage is much lower.

Positive contrast media are the most suitable for studying the entire spinal canal, assuming, of course, that the cerebrospinal fluid pathways are clear. When there is a block, myelography should be repeated, either above or below the obstacle. In our series, block of the cerebrospinal pathways was encountered in 15 of the patients with disk prolapse and in 2 patients of the other groups.

SUMMARY

1. Out of 952 patients operated on for spinal cord lesions, 100 or 10.50 per cent had multiple lesions.

2. This is a higher incidence than is generally assumed.

3. Most of the multiple lesions (82 per cent) occurred in disk prolapse cases, 14 per cent were found in patients with tumors and 4 per cent in the miscellaneous group.

4. Bone changes in several sites of the spine were observed in only 6 cases.

5. Myelography of the entire spinal canal is the procedure of choice in the diagnosis of these multiple lesions. It is also an aid in clarifying the atypical symptoms.

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BONE ISLAND*

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A SOLITARY, dense, asymptomatic nodule demonstrated on roentgen studies of bone is most frequently referred to as a "bone island." Although it is not a rare finding, little is reported in the literature regarding its histology or origin. It has been accepted as a benign lesion and of no clinical significance. Some of the lack of information regarding this phenomenon is manifest in the various names used: "focal sclerosis" by Caffey,[1] "calcific island in bone" by Steel,[5] and "sclerotic bone island" by Meschan.[3] It is the purpose of the authors to help clarify the origin as well as the histologic pattern of this lesion.

REPORT OF A CASE

Case 1 (U-2468). This 75 year old white male was admitted following a stroke. An abdominal roentgenogram showed a bone island in the left iliac wing (Fig. 1*A*). The physical examination and laboratory data were all compatible with his neurologic disease. There had never been any symptoms referable to the area of the bone island. The patient expired as a sequela of the stroke. At autopsy the bone island was removed *in situ* with the entire thickness of iliac bone (Fig. 1*B*).

Roentgen Findings (Fig. 1*A*). A homogeneously dense, 1 cm. in diameter, nodule was demonstrated in the left ilium. The margin of the nodule had a thorny radiation which intermingled with the trabeculae of the medullary bone. This nodule was present 4 years previously with identical findings.

Gross Findings (Fig. 1*B*). The cut surface was that of cortical bone. It was hard and yellowish white. The nodule lay in the medulla and was partially connected with the anterior cortex.

Microscopic Findings (Fig. 1, *C* and *D*). The nodule had a haversian system of canals, lamellae, and lacunae indicating compact bone (Fig. 1*D*). Part of the island was a continuation of the cortex. The rest of the nodule was connected to the architecture of the medullary bone with thickened trabeculae which produced the thorn-like projections of the island visualized on the roentgenogram. The island was old bone without evidence of new bone formation. No osteoclasts were seen. The medullary space at the region of the island was more cellular and less fatty than normal marrow space. No cartilage or fibrous tissue was seen.

DISCUSSION

The bone island is not a calcification of cartilage or of fibrous tissue. It is solid bone. Close observation of 42 bone islands revealed in most of them a uniformly com-

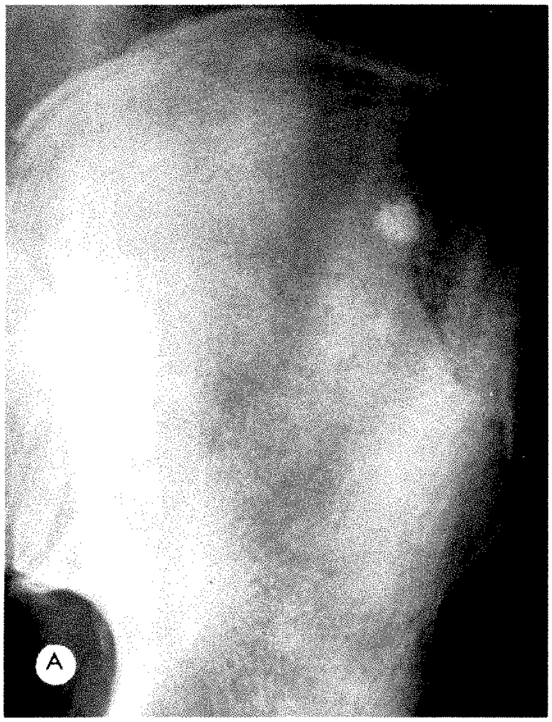

Fig. 1. Case 1. (*A*) Iliac bone island shows homogeneous density and radiating thorny trabeculae at the margin.

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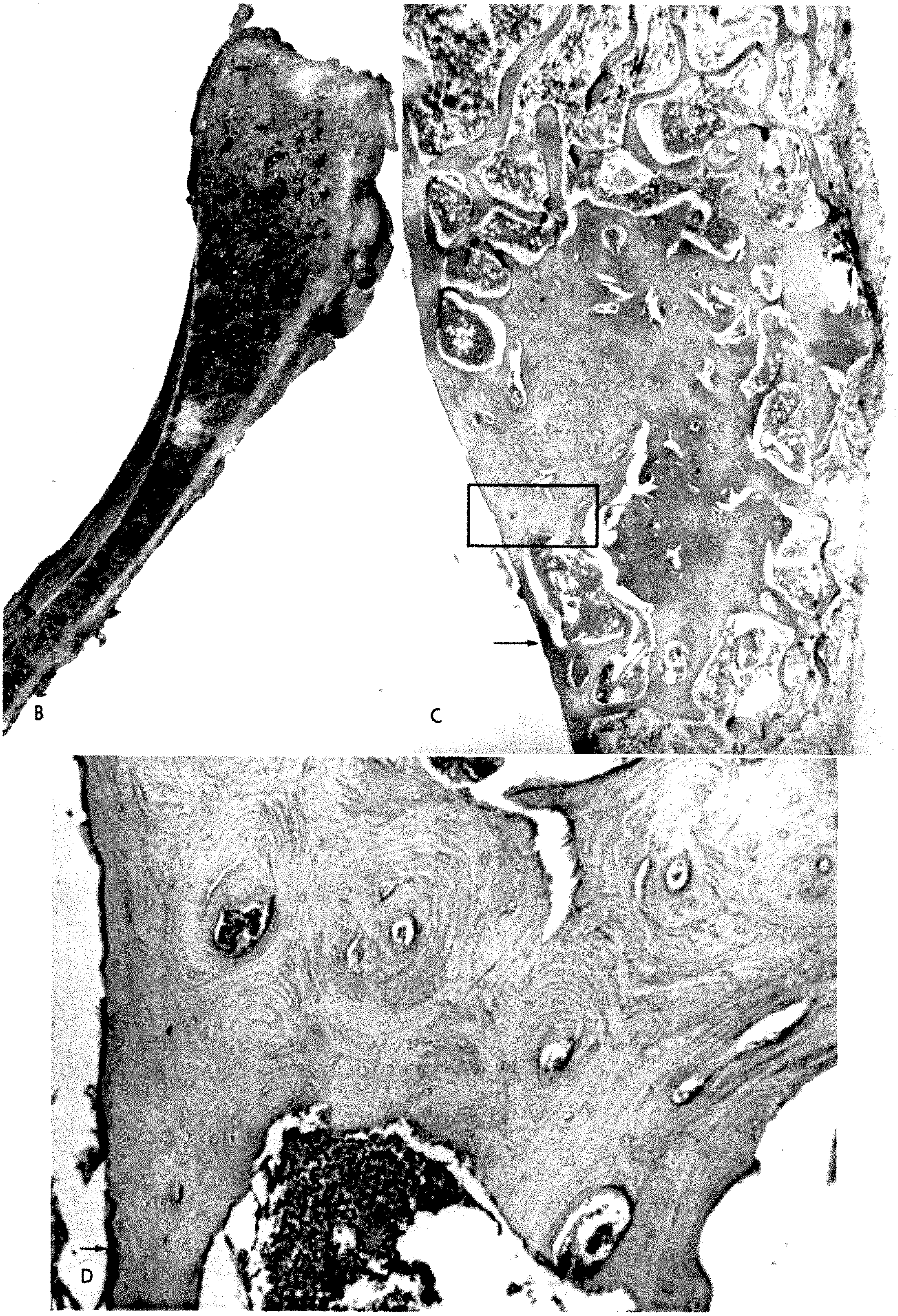
B
C
D

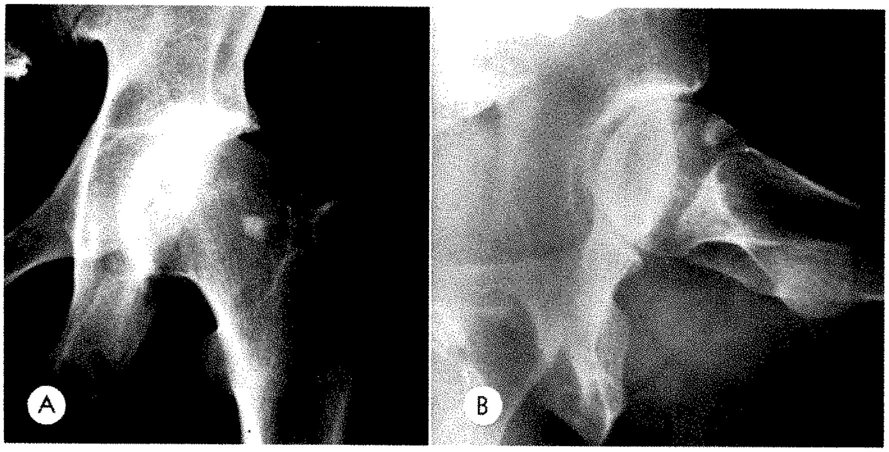

FIG. 2. (*A* and *B*) An oval left femoral neck bone island lies under the cortex and along the axis of the main trabecular architecture.

pact bone density with peripheral radiation of trabeculae. Schmorl[4] described osteopoikilosis in detail and included a microscopic study in his paper in 1931. The histology, location, and roentgenographic findings of the bone island are similar to those of osteopoikilosis except that one is solitary and the other is multiple. Steel[5] reported the microscopic findings of a bone island from the femoral neck similar to that seen in Figure 2, *A* and *B*. It showed compact bone and ossifying cartilage. No calcification of degenerative tissue was noted. Ferguson[2] described a calcified medullary defect. It was composed of amorphous calcification of degenerated cartilage and was not a bone island. These calcified medullary defects showed an aggregation of mottled irregular calcification on roentgenograms.

ETIOLOGY

The etiology of the bone island is not known. It does not relate to any disease or trauma. It appears that the island is developmental. Probably localized excess bone formation occurs in the trabecula of spongiosa or at the medullary side of the cortex in the life of normal bone which is constantly being resorbed and reformed. No new bone island has been reported which had not been seen previously. The island is stable for years. One case was observed for 18 years and remained unchanged

INCIDENCE

Bone islands are not rare in adults. They are less frequently seen in pediatric radiology. There has been no sex difference noted.

DISTRIBUTION

In a series of 5,000 consecutive patients having some type of roentgenologic examination which included bony structures, 42 bone islands were noted. They were distributed as follows:

←

FIG. 1. (*B*) Gross specimen. The dense compact bone island lies in the spongiosa and close to the anterior cortex. (*C*) Microscopic examination shows that the bone island is a continuation of anterior cortex (arrow) of the iliac bone. The medullary side of the bone island is connected with the thickened trabeculae of the spongiosa which on roentgenograms had the appearance of thorny radiations. The marrow space is more cellular at the region of the bone island. No cartilage or fibrous tissue is seen. (*D*) High power view of the bone island from blocked area of *C*. The normal histology of the compact bone is seen. The haversian system of canals, lamellae and lacunae is well demonstrated (arrow—anterior cortex).

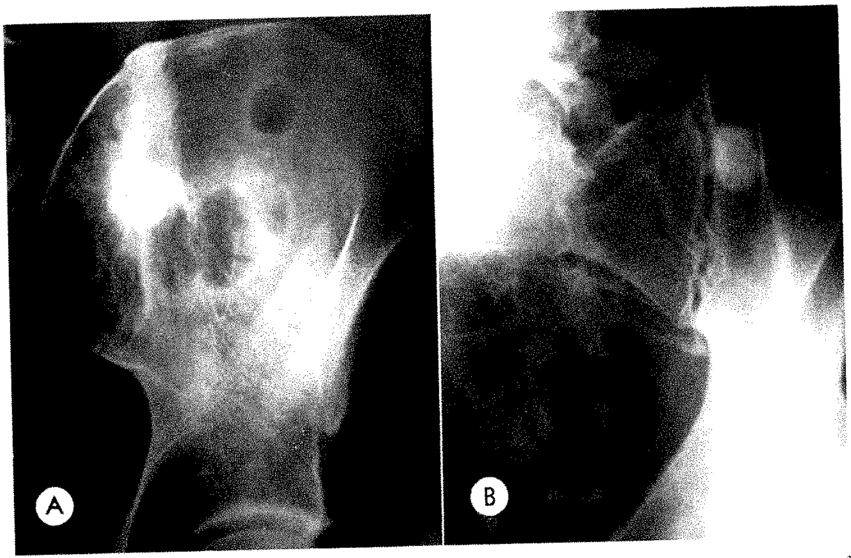

Fig. 3. (*A* and *B*) The iliac bone island is juxta-articular in position. The island shows areas of thorny radiations and a sharp margin. The bone island often overlies the sacrum but seldom is found in the sacrum. Its location is in the ilium, near the articular cortex.

Ilium		25
Wing Area	15	
Acetabular Area	10	
Femur		12
Neck and Head	11	
Trochanters	1	
Metatarsus (end)		1
Spine (spinous process)		1
Rib		1
Tibia (proximal)		1
Humerus (head)		1
Total		42

These lesions have been reported in all bones except the skull. The frequent involvement of the bone about the hip is similar to osteopoikilosis.

ROENTGEN FINDINGS

Ordinarily, the island is ovoid or oblong in shape and runs along the bony axis of the trabecular architecture (Fig. 2, *A* and *B*; 4; and 5). The largest one in this group measured 2.5×1.5 cm. It is homogeneously dense. The margins of the island show radiating bony streaks which intermingle with trabeculae of spongiosa (Fig. 1; 3, *A* and *B*; 4; and 5). Some of the streaks run in bunches, forming a brush effect (Fig. 4). Part of the margin may have a sharp demarcation against relatively lucent spongiosa. The density of the island depends on

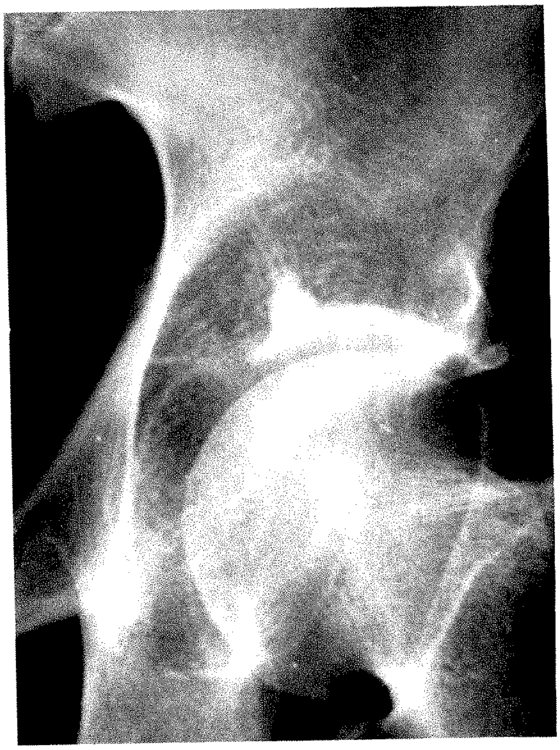

Fig. 4. Juxta-articular or cortical position of the island. Streaks of radiating trabeculae run along the main architecture of the spongiosa and form a brush effect.

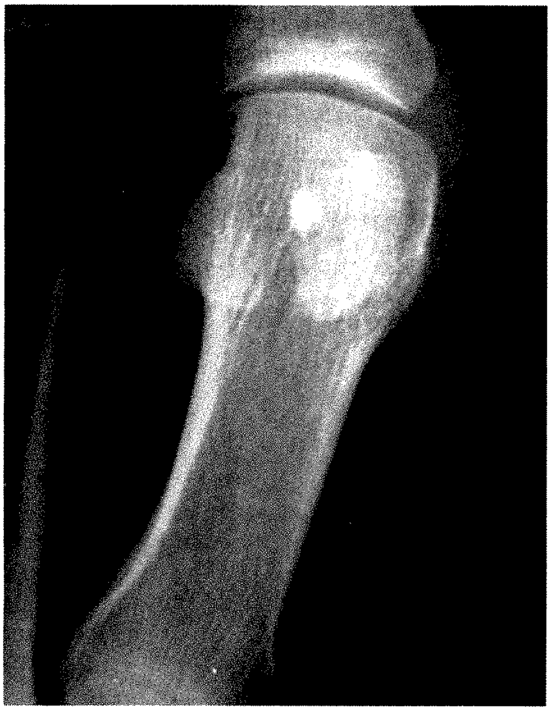

FIG. 5. Metatarsal bone island has thorny radiating trabeculae which intermingle with the trabeculae of the spongiosa. Also, bone islands run along the axis of the main trabeculae.

the amount of compact bone. Some of the islands are less dense, with an appearance similar to that of an osteoblastic metastatic nodule in bone.

DIFFERENTIAL DIAGNOSIS

1. *Osteoblastic metastasis.* An osteoblastic metastatic nodule is usually less dense and has a granular appearance. The margin of the metastatic nodule does not show the

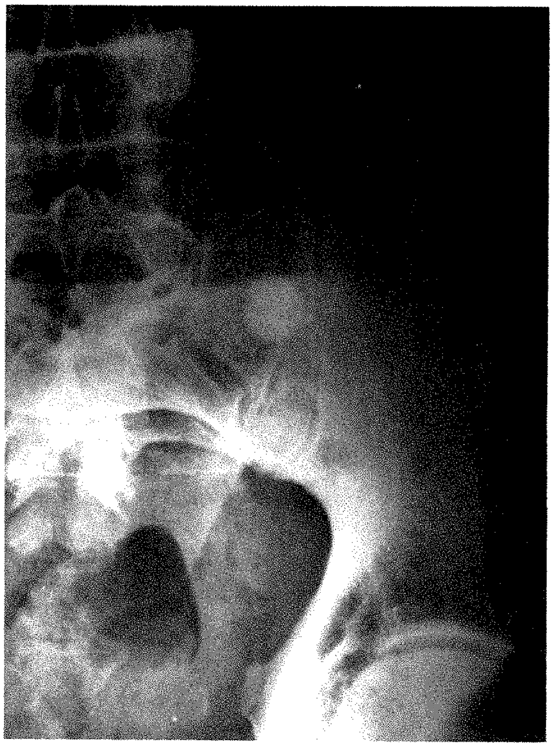

FIG. 7. This is an osteoblastic metastasis of prostate cancer in the ilium adjacent to the joint (not in the sacrum). This solitary metastatic nodule remained unchanged for 4 years. It was not present previously. The nodule is less dense and granular. Also, no thorny radiations at the margin are noted.

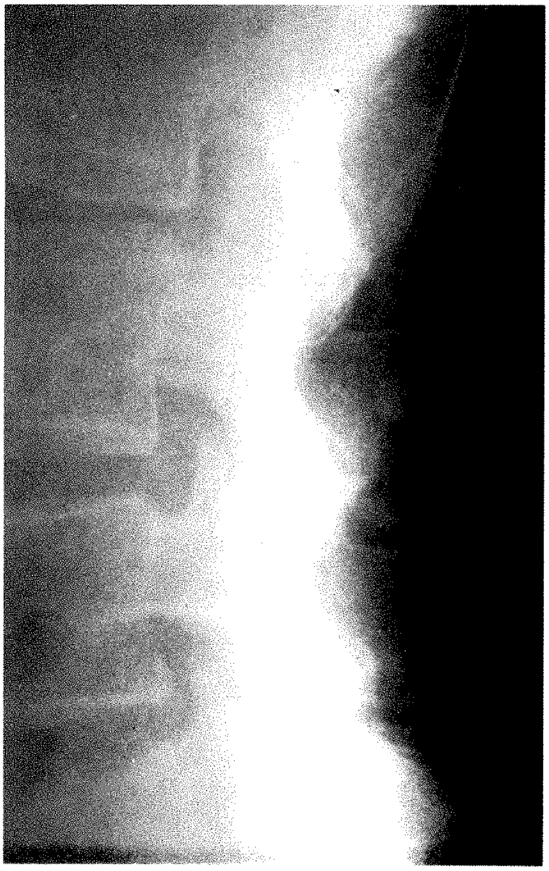

FIG. 6. Bone island of spinous process of the lumbar spine.

radiating streaks or the sharp margin of that of a bone island. However, occasionally they are indistinguishable from one another, especially when a bone island has less density. A case of solitary prostatic cancer metastasis to the ilium (Fig. 7) has been followed for 4 years without alteration in configuration or density while the patient has been undergoing hormone therapy for the cancer. The roentgen appearance is granular without radiating streaks at the margins.

2. *Medullary calcification.* This is not a bone island. As Ferguson stated, it is calcification of degenerated cartilage or fibrous arrest in the spongiosa and on roentgenograms a mottled irregular calcific shadow without the solid uniform density of a bone island is visualized. Common sites of such medullary calcifications are the distal femur and proximal tibia. The roentgenographic appearance of a bone infarction is similar to that of the medullary calcification.

SUMMARY

1. An autopsy study of a bone island is described. The roentgenographic findings of 42 bone islands are discussed.

2. A bone island is compact bone. The nature of the island is similar to that of osteopoikilosis except that it is solitary. The island has radiating trabeculae at the margin.

3. Medullary calcification is a calcification of degenerated tissue, and not a bone island.

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THE FRACTURED ULNAR STYLOID

ROENTGEN DIAGNOSIS OF RECENT ISOLATED SPRAIN FRACTURE

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FRACTURES of the styloid process of the ulna occur most frequently as an associated injury with the classic Colles' fracture. They are present in about 50 per cent of these cases and are the result of direct violence from a fall on the outstretched hand.[1,5] Occasionally, fractures of the styloid may occur as a separate injury from indirect violence due to a sudden forceful radial twist of the wrist. The whole or a part of the process is avulsed from the strain on the ulnar collateral ligament and the roentgenogram shows only this injury without any other lesion. The information concerning the roentgenographic features of these isolated sprain fractures is meager. Without a knowledge of these features, the patient may be inadequately treated and possibly later subjected to amputation of the styloid,[2] which in turn is followed by a permanently weak wrist.[4] A typical case is presented and the features most likely to be of interest to the roentgenologist are outlined.

REPORT OF A CASE

On January 2, 1958, an 18 year old white male laborer in a junk yard, had roentgenograms made of his right wrist for the possible presence of a foreign body. These showed no evidence of significant abnormality (Fig. 1, *A-D*).

On January 9, 1958, one week after his roentgenographic examination, he forcefully twisted his right wrist towards the radial aspect while loading scrap metal on a truck. After this, there was annoying discomfort and weakness in the wrist but not of sufficient degree to prevent working. The same day he visited his physician who found slight tenderness over the styloid of the ulna but otherwise nothing of significance. There was no evidence of soft tissue swelling or disturbance of function. A diagnosis of strain was made, novocaine was injected about the styloid and an elastoplast bandage was applied. He was permitted to continue work. The annoying discomfort persisted.

On January 12, 1958 he again visited his physician, received a second injection of novocaine and reapplication of the elastoplast bandage.

On January 15, 1958, six days after the injury during all of which time he continued working, additional roentgenograms were obtained since no improvement in the patient's condition was noted. These showed what appeared to be an old pseudoarthrotic healed fracture of the ulnar styloid or an anomalous hypoplastic styloid with an os styloides or os triangulare (Fig. 2, *A-D*). The fragment of styloid, as well as the ulna in the neighborhood of where the styloid had been attached, was completely covered by a thin layer of dense bone with all bounding margins smooth and rounded. Multiple views disclosed no definite defect in the ulna from which the styloid fragment was derived. The jagged sharply angular margins at the fracture surfaces such as one would anticipate with a recent fracture were not demonstrated and there was no overlying soft tissue swelling.

On January 15, 1958 the hand, wrist and forearm were enclosed in an unpadded plaster cast. After 4 weeks, the cast was removed and the patient was advised to avoid straining the wrist for a period of at least 4 weeks longer. Recovery was uneventful and regular work was resumed 8 weeks following the date of injury. Roentgenograms obtained 4 and 10 weeks after the first ones showed no significnat changes in the appearance of the bones.

CLINICAL SIGNS AND SYMPTOMS

The clinical signs and symptoms of these fractures, as exemplified by the above case, present a distinctly different pattern from those usually seen in fractures elsewhere: (1) there is relatively little pain, the feeling being rather an annoying discomfort; (2) there is only slight disability or limitation

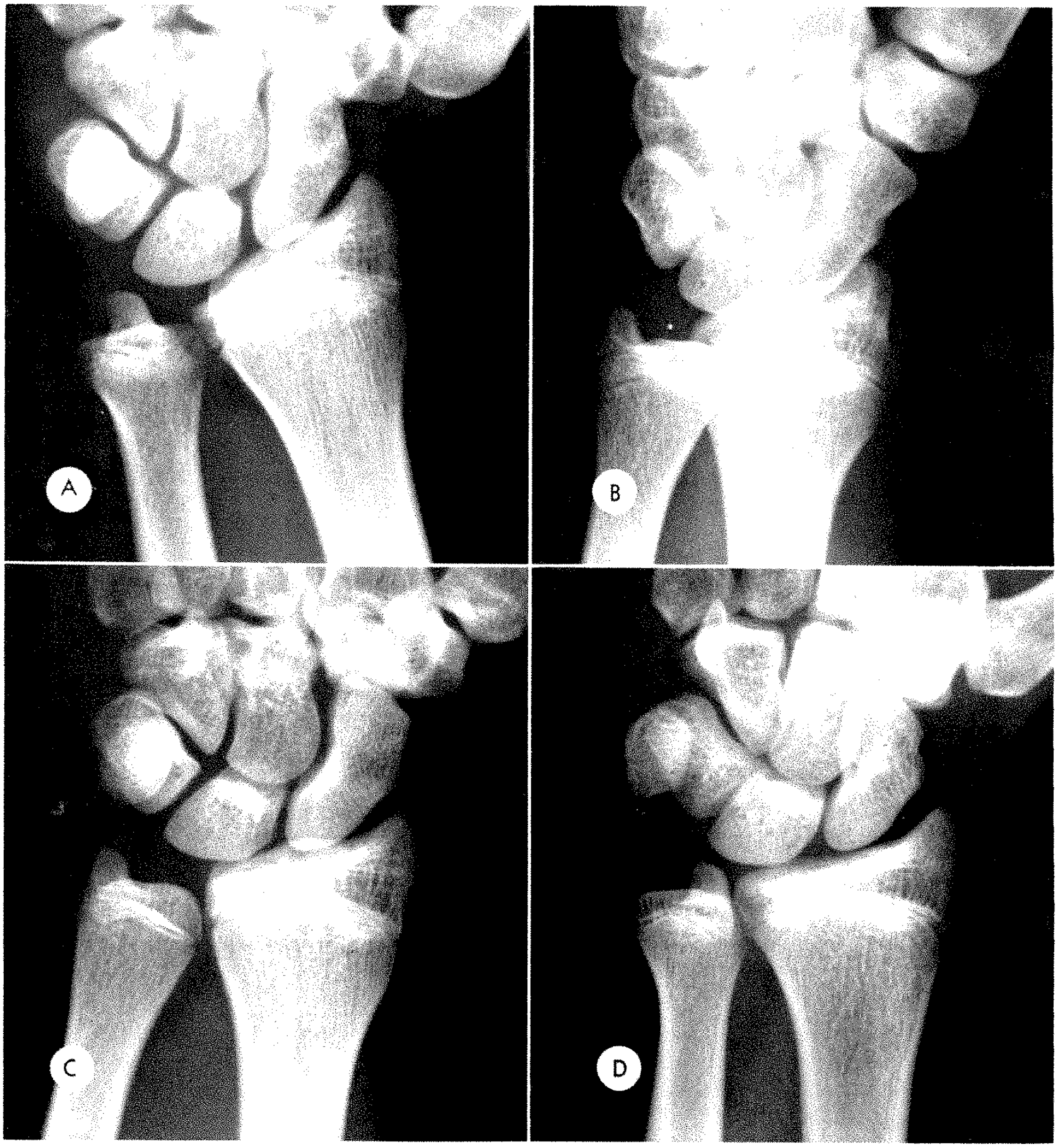

Fig. 1. (*A–D*) Roentgenograms of right wrist obtained 1 week before injury show no evidence of bone or joint abnormality. Note the firm bony attachment of the styloid on the distal epiphysis of the ulna. In spite of the firm attachment, however, a forceful radial twist of the wrist can transmit sufficient stress to the attached ulnar collateral ligament as to completely avulse the process.

of function; (3) discomfort and dysfunction are insufficient to prevent extensive use of the wrist; (4) there is little or no soft tissue swelling; and (5) pressure by palpation over the fracture reveals only slight discomfort in contrast to the marked degree of tenderness customarily found at the site of fractures. These are the signs and symptoms to be expected from a strain or slight sprain of the wrist rather than from a fracture.

ROENTGEN DIAGNOSIS

In the case reported above, the roentgenograms obtained 1 week before injury showed a normal wrist. Roentgenograms obtained 6 days after the injury disclosed

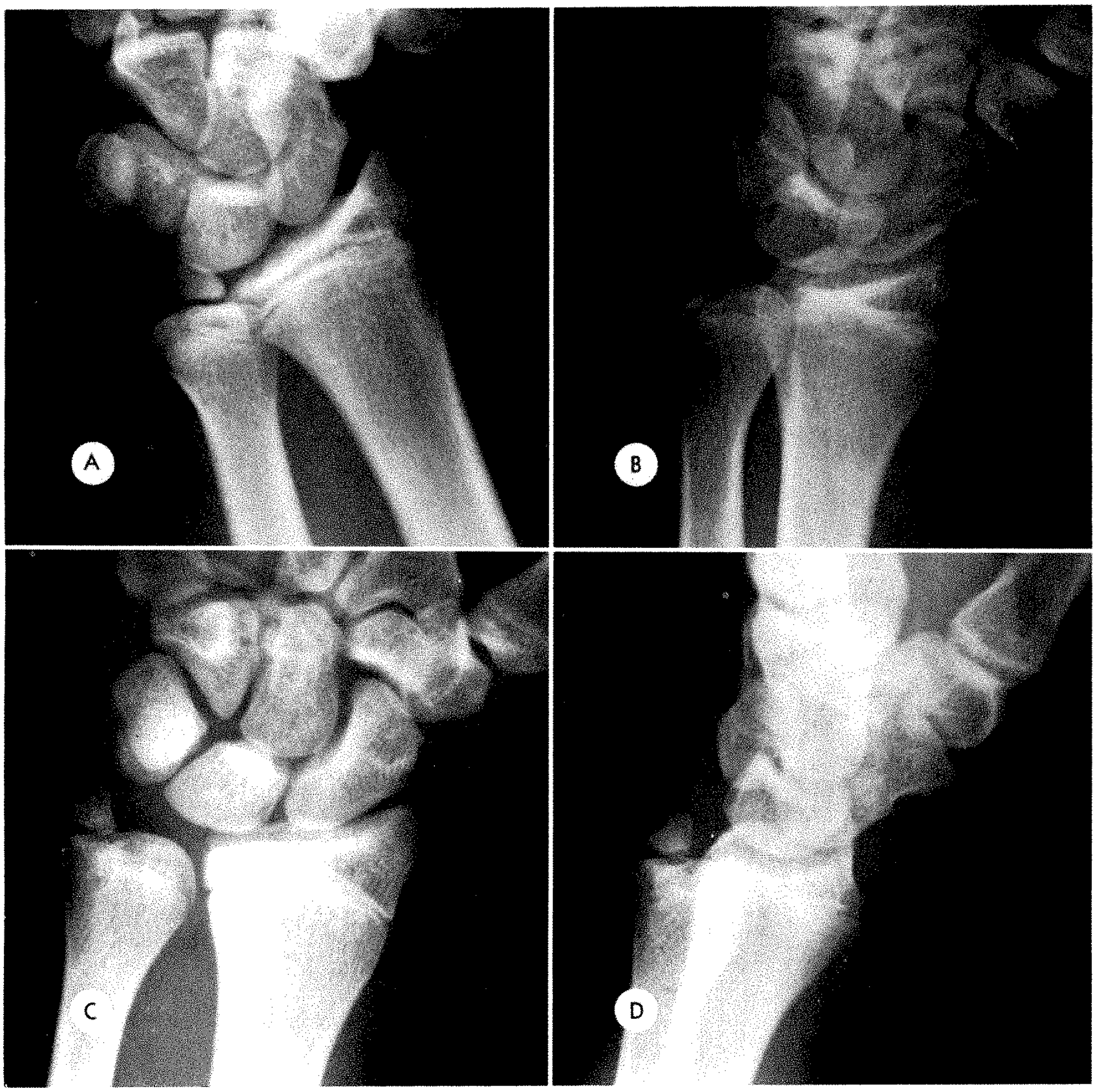

Fig. 2. (*A–D*) Roentgenograms of right wrist obtained 6 days after the injury show what appears to be an old pseudoarthrotic healed fracture of the ulnar styloid or an anomalous hypoplastic styloid with an os styloides or os triangulare. Note the absence of jagged sharply angular margins at the fracture surfaces such as would be expected with a recent fracture. Roentgenographic evidence of soft tissue swelling is absent.

the typical appearance of an old fracture or a possible anomaly. Due to the relatively slight discomfort which these patients suffer, there is little or no limitation in the use of the injured wrist so that the jagged sharply angular margins of the fracture become worn, round and perfectly smooth. The defects in the ulna from which the styloid fragments are derived, as well as the bare fracture surfaces of the fragments, become bounded by a fine dense smooth layer of bone, preventing detection of the sites where the bones once fitted into one another. The rapidity with which these changes take place depends upon the degree of use of the wrist: the greater the amount of use, the earlier the changes. Thus, within an amazingly short period of time these injuries may acquire the appearance of an old pseudoarthrotic healed fracture of the ulnar styloid, or of an anomalous hypoplastic styloid with an os styloides or an os triangulare. As a rule, there is little or no soft tissue swelling detectable on the roentgenogram.

ROENTGEN DIFFERENTIAL DIAGNOSIS

If, as in the case reported above, roentgenograms obtained shortly before the accident are present for comparison, the differentiation of recent fracture from an old pseudoarthrotic healed fracture or an anomalous styloid with sesamoid is simplified. In most cases, however, these are not likely to be present and the roentgenogram must be searched for evidence of an old injury, especially for residual deformity from an old Colles' fracture, or inquiry must be made as to whether there has been treatment for Colles' fracture in the past. A positive history would suggest an old injury.

Anomalous hypoplastic styloid with os styloides or os triangulare is a rare anomaly which is likely to be bilateral.[3,7] Roentgenograms of the uninjured wrist would thus assist to exclude this possibility. Undoubtedly, cases described as anomalous hypoplastic styloids with os styloides or os triangulare have in many instances been cases of old fractured styloids healed by fibrous union,[3,7] a residue present in about 25 per cent of the cases of Colles' fracture.[1]

Other conditions which may appear about the styloid, such as vascular calcifications distinguished by their tendency to outline a tube, granular or lumpy calcifications in bursa, lozenge-shaped calcifications in the discus articularis and osteoarthritic deposits, are usually readily differentiated from fracture and require no mention here.[7]

TREATMENT

Injury to the ulnar styloid following a forceful twist of the wrist accompanied by undiminishing tenderness and discomfort, and which on roentgenograms obtained 5 to 10 days later appears to represent an old pseudoarthrotic healed fracture or an anomalous hypoplastic styloid with sesamoid, should be regarded and treated as a recent sprain fracture. This indicates that the forearm, wrist and hand should be enclosed in an unpadded plaster cast until firm union has taken place, usually a period of 4 to 12 weeks.[1,6] Healing occurs mostly by fibrous union but a painless strong wrist regularly results.[1]

If, on the other hand, an incorrect diagnosis of strain or simple sprain is made and the patient is treated with strappings, novocaine injections, baking, massage and ultrasound, the discomfort in the wrist is likely to persist unabated for months. Finally, in order to bring relief, the styloid, a necessary structure for the stability of the wrist, is amputated.[2] This may remedy the discomfort but it does so at the expense of a permanently weak wrist through loss of the insertion of the ulnar collateral ligament.[4]

SUMMARY

Due to the mild nature of the symptoms from recent sprain fracture of the ulnar styloid, not infrequently a period of 5 to 10 days from the time of injury elapses before roentgenograms are obtained. These roentgenograms may show what appears to be an old healed fracture of the styloid or an anomalous hypoplastic styloid with sesamoid. Absence of roentgenologic evidence of overlying soft tissue swelling and the clinical symptoms support a diagnosis of a trivial injury, such as a strain or mild sprain, instead of a serious recent sprain fracture. Treatment for sprain instead of fracture may lead to a long period of discomfort in the wrist for the relief of which amputation of the styloid may be advised. This operation leads to a permanently weak wrist.

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CAMPTODACTYLY*

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THE aim of the present authors is to report the clinical history and findings of 19 new patients with camptodactyly (Table 1) and to review the main features of this disorder.

DEFINITION

Camptodactyly (from kamptein, to bend and dactylos, finger) is a special type of permanent flexion contracture of fingers, which may be congenital or acquired, sporadic or genetically determined. The flexion deformity is always located at the proximal interphalangeal joint. It involves the little finger, often the ring finger, and occasionally the middle and index fingers as well. Its pathologic anatomy is only partially known and its etiology is obscure. The disorder is uncommon but not rare.

HISTORY AND SYNONYMS

Although mention of the disorder can be found by earlier writers,[3,15,38] it was Landouzy and Adams who first described this type of finger contracture in detail. Landouzy used the term camptodactyly in a clinical lecture delivered in 1885,[17] and in an article published in 1906.[16] Adams[1] described the condition even in greater detail in 1891 using the term of congenital contracture of the fingers; his communication is still one of the most informative on the subject. Several articles on the disorder have appeared subsequently, most of which are listed in the bibliography at the end of this paper.

In addition to the terms already mentioned, the anomaly has also been referred to as streblomicrodactyly (from streblos, crooked, and micros dactylos, little finger), campylodactyly (from kampylos, curved, and dactylos, finger), palmar clinodactyly (from klinein, to bend, and dactylos, finger) congenital Dupuytren's contracture, and congenital, familial, hereditary crooked, flexed, or hammer (little) fingers.

CLINICAL FINDINGS (Fig. 1–5)

The flexion contracture of the finger is characteristically located at the proximal interphalangeal joint, but a milder degree of flexion deformity at the distal interphalangeal articulation may co-exist. The metacarpal phalangeal articulation is often hyperextended, never flexed.

The little finger is involved in practically all instances. In more than half of the cases, this is the only finger affected. In addition to being flexed, the little finger may also be rotated with its palmar surface facing the other fingers (Fig. 4, *A–D*). An associated flexion contracture of the ring finger occurs in one-fourth to one-third of the cases. Occasionally, the middle and rarely the index fingers are also affected. Involvement of the thumb does not occur or is very rare. When the contracture is present in more than one finger, usually all the more medially placed fingers are affected, without skipping of fingers.

Almost invariably the little finger is the most severely affected. When more than one finger is involved, the severity of the contracture usually decreases progressively from the little to the index finger.

Camptodactyly is bilateral in 60 to 70 per cent of the cases. When unilateral, the contracture is apparently more frequent on the right; when bilateral, it tends to be symmetric in distribution but is often more severe on the right than the left.

The degree of flexion varies from mild to severe. A flexion greater than 90° is uncommon. Minimal degrees of flexion of little fingers at the proximal interphalangeal joint are relative frequent findings in adults and probably should not be considered pathologic.

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TABLE I*

Patient No.	Sex	Age When Examined	Anomaly First Noted	Finger Involved	Soft Tissue Changes	Course	Symptoms	Treatment and Results	Familial History	Associated Congenital Anomalies
1	M	1 mo. and 10 yr.	Birth	Both little fingers	± at 1 mo.; + at 10 yr.	Deformity became noncorrectable passively sometime between 1 mo. and 10 yr.	None	Extension splint in early infancy; no improvement	No	Right strabismus
2	F	19 and 21 yr.	Birth	Little and ring fingers bilaterally	+	Stationary	None	None	Sisters	Ventricular septal defect, web-neck, bilateral ptosis, strabismus, pectus excavatum. Turner's syndrome (?)
3	F	23 yr.	"Early life"	Right little finger	+	Stationary	Slight interference with typing	None		None
4	F	31 yr.	16 yr.	Little and ring fingers bilaterally (Fig. 1, *A–D*)	+ (but normal creases and no skin web)	?	None	None	No	None
5	F	14 yr.	?	Both little fingers	+	?	None	None	No	Slight mental retardation
6	M	17 and 30 yr.	?	Right little, ring and middle fingers; all 4 fingers on left (Fig. 2, *A* and *B*)	+	?	None	Capsulotomy, with lengthening of flexor tendons and skin followed by prolonged extension in cast (right little and ring fingers) at age 17 years; 1 year later: ankylosis	?	Hammer toe deformity of right great toe and of both third toes, right supranumerary nipple, left hemithorax larger than right
7	M	9 yr.	Birth	All 4 fingers bilaterally (Fig. 3, *A* and *B*)	+	?	None	Capsulotomy and skin lengthening (left little finger) at 9 years; no improvement	No	Mild varus deformity of left foot
8	F	6 mo. and 7 yr.	Birth	Little and ring finger bilaterally (Fig. 4, *A–E*)	? at 6 mo.; + at 7 yr.	Deformity became noncorrectable passively between 6 mo. and 7 yr.	None	None	No	Club feet, bilateral ptosis, left ureteropelvic junction obstruction. Pituitary dwarfism (?)
9	M	13 and 29 yr.	Birth	Right little finger	+	Stationary between 13 and 29 yr.	None	Skin lengthening at 13 years; no improvement	?	None
10	F	51 yr.	?	Left little finger	?	?	?	None	?	None
11	M	10 wk.	Birth	Both little fingers	?	—	—	Extension splints in infancy; no improvement	?	Mental retardation
12	M	Birth	Birth	Both little fingers	?	—	—	None	?	Hydrocephalus, imperforate anus, club feet, absence of right pectoral muscle
13	M	18 yr.	Birth	Both little fingers	+	?	None	None	No	Kleinfelter syndrome
14	M	14 mo. and 7 yr.	Birth	Both little fingers	? at 4 mo.; + at 7 yr.	Deformity became noncorrectable passively at approximately 1 yr.	None	Passive stretching in infancy; no improvement	No	Marfan's syndrome
15	F	25 yr.	"Early life"	Both little fingers	+	?	?	None	No	None
16	M	19 yr.	?	Both little fingers	+	?	?	None	No	None
17	M	6 mo.	Birth	Left little finger	+	Deformity noncorrectable passively at 6 mo.	—	Passive stretching in early infancy; no improvement	No	None
18	F	11 and 12 yr.	Birth	Little and ring fingers bilaterally (Fig. 5, *A* and *B*)	+	?	None	Incision of subcutaneous fibrotic bands and skin lengthening, followed by prolonged extension in cast at 11 years; slightly worse 1 year later	?	None
19	M	50 yr.	?	Right little finger	+	?	?	None	?	None

* *Additional data:* All patients were white except 3 (Patient 5, 7 and 18) who were Negroes. In all patients the flexion deformity of the fingers was obvious even in the mildest cases. The soft tissue changes in column 6 refer to a web-like shortening of the skin, absence or poor definition of the transverse creases, and tight subcutaneous bands and deeper soft tissues, all in the volar aspect of the affected fingers. None of the patients had any neurologic abnormality, previous infections or trauma to the hands or fingers, or other disorders which could explain the contracture. Roentgenograms of the affected fingers were made in all patients; all the available lateral roentgenograms of the affected little fingers in this series of patients are shown in Figure 6, *A–D* and Figure 7, *A–E*. The findings at operation described in the 4 patients who had surgery were as follows: in Patient 6 there was a contracture of the skin, flexor tendons and articular capsule; in Patient 7 there was a contracture of the skin and articular capsule (status of other deep structures not mentioned); in Patient 9 the skin and subcutaneous tissue were shortened (status of deep structures not mentioned); in Patient 18 the skin was contracted and tense longitudinally placed fibrotic bands were present beneath the skin, the lateral ligaments were shortened and the flexor tendons and sheaths were "apparently normal."

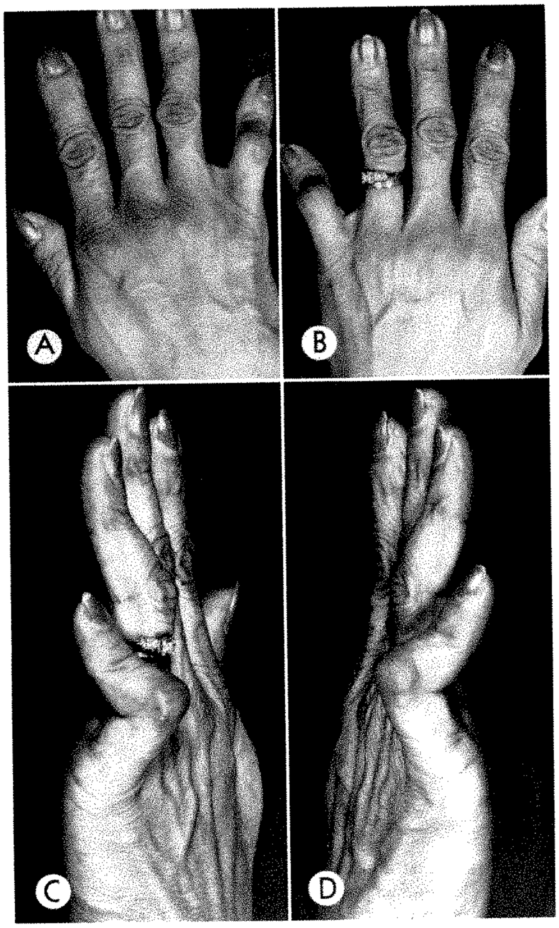

Fig. 1. Patient 4. (*A–D*) Photographs of hands at 31 years of age.

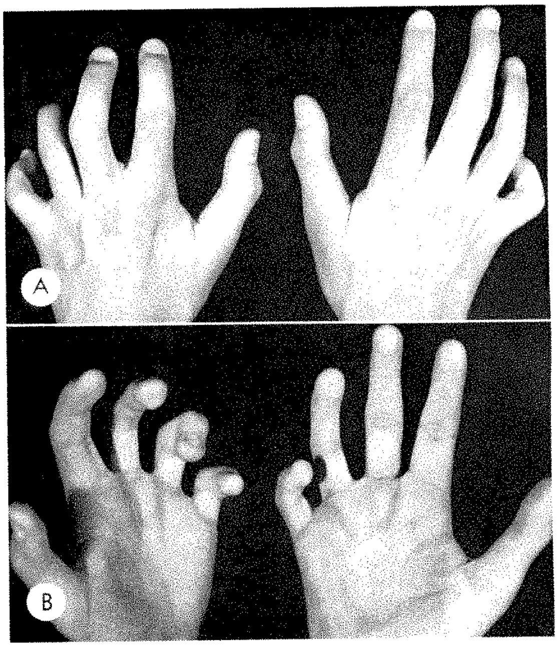

Fig. 2. Patient 6. (*A* and *B*) Photographs of hands at 17 years of age.

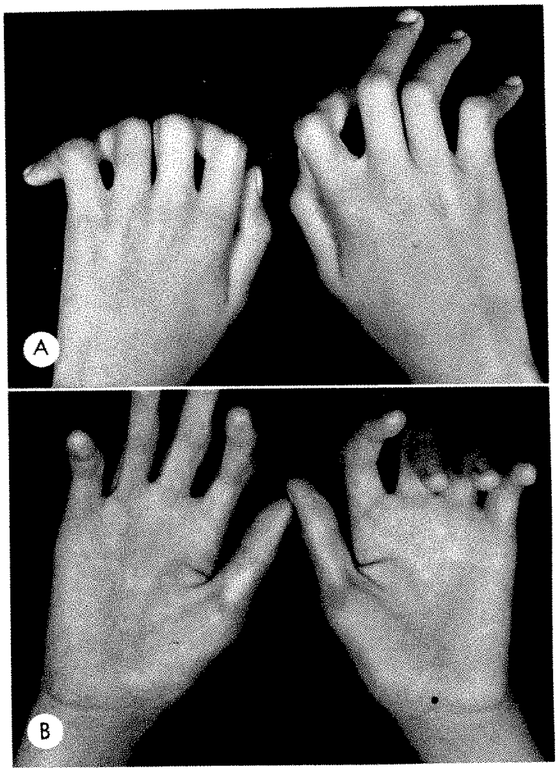

Fig. 3. Patient 7. (*A* and *B*) Photographs of hands at 9 years of age.

Active straightening of the affected fingers is never possible. Passive straightening is usually impossible when the disorder is brought to medical attention. Further active and passive flexion is usually not hindered.

The affected finger is more gracile than normal. The skin of its palmar surface tends to be thin and is always quite smooth without puckerings or ridges; it is often shortened and may resemble a longitudinal web extending from the proximal to the middle or distal phalanx. Its normal transverse creases at the level of the joints are often poorly defined and may be completely obliterated. On palpation, the deep soft tissues of the flexor aspect of the bent fingers commonly feel tense and short, and

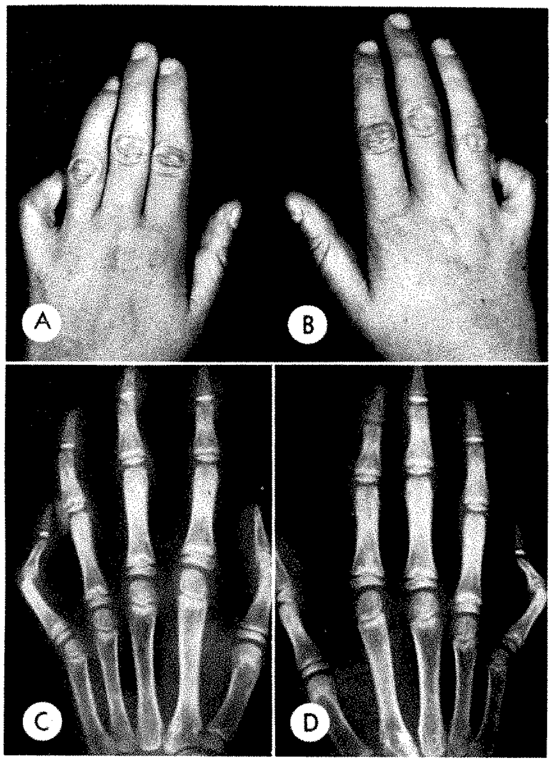

Fig. 4. Patient 8. (*A* and *B*) Photographs and (*C* and *D*) roentgenograms of hands at 7 years of age.

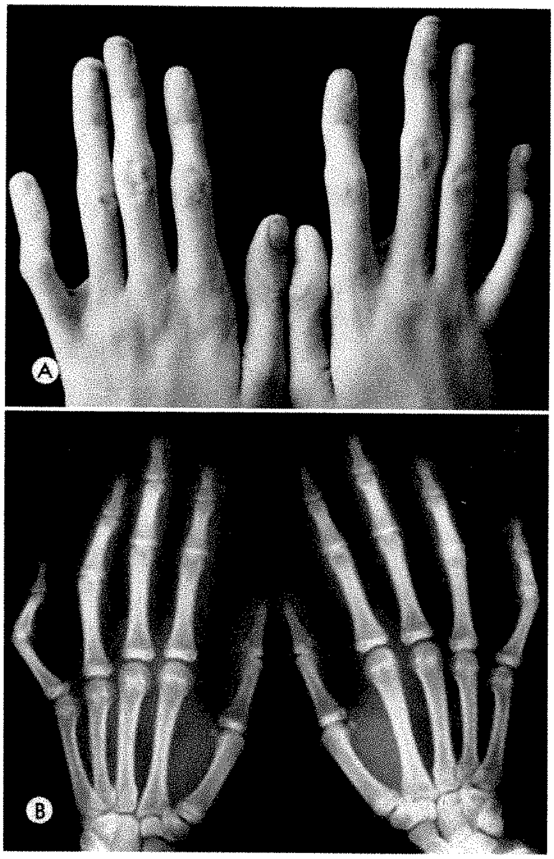

Fig. 5. Patient 18. (*A*) Photograph and (*B*) roentgenogram of hands at 11 years of age.

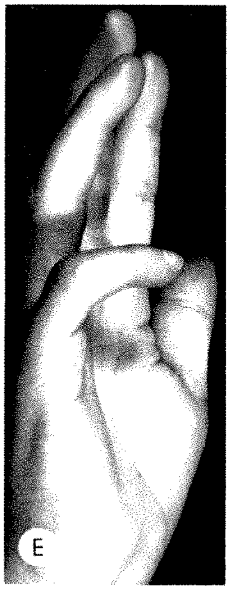

Fig. 4. (*E*) Lateral photograph of hand at 7 years of age.

tense cord-like bands running longitudinally from the first to the second phalanx may be felt underneath the skin. All these soft tissue changes are especially evident when passive straightening of the finger is attempted. The soft tissues of the rest of the hand appear normal, and there are no demonstrable neurologic abnormalities in the arm, hand or fingers.

ONSET AND COURSE

The disorder may be congenital or, less commonly, acquired. The congenital form may be discovered at birth or shortly thereafter. The acquired form may have its onset during childhood or more often in adolescence.

The course of the acquired form of the disease is not known. That of the congenital form has been divided by Adams[1] into 3 stages. In the *first stage* there is only a "dropping" of the last two phalanges of the little finger which may also be rotated toward the median line of the hand. The skin and deep soft tissues of the affected finger are still normal and the deformity can still be corrected passively. The *second stage* is essentially one of confirmed contracture. It is reached gradually, seldom before the seventh to tenth year of life. Passive straightening of the fingers is no longer possible. The proximal phalanx is commonly hyperextended. The soft tissue changes described above are now evident. Spontaneous arrest of the contracture may occur in the second and sometimes even in the first stage. In a few cases a *third stage* is reached between the fifteenth and twentieth year of life. During this stage the flexion contracture increases and spreads to other fingers, the changes in the skin and deeper soft tissues become more pronounced and the hand becomes less and less useful. No further progression occurs after the twentieth year.

This course of the disorder, described by Adams[1] and generally accepted by the subsequent authors, is far from constant. The deformity may remain stationary throughout life. Multiple finger involvement, irreducible contracture, and well developed soft tissue changes may already be present at birth or in very early childhood. Severe long-standing flexion deformity may occur without obvious contracture of the skin or loss of the transverse cutaneous creases.

SYMPTOMS

Usually, the deformity is of little or no clinical significance, but in some cases, particularly when severe or when several fingers are involved, it may interfere with the functional use of the hand as in typing, piano playing and other activities, or it may be troublesome to the patient for cosmetic reasons. There is no pain or sign of local inflammation at any time during the course of the disease.

SEX INCIDENCE AND GENETICS

There is evidence to suggest that females predominate in the acquired form, while the congenital form apparently occurs with an approximately equal frequency in both sexes.

The anomaly may be sporadic or familial. The familial form has received more attention in the literature than the sporadic but is definitely less common. The mode of transmission of the inherited cases is estimated to be that of a simple dominant characteristic with variable penetrance and expressivity. Familial and hereditary instances have been reported both in congenital and acquired cases.

ASSOCIATED ANOMALIES

Several authors have remarked on the frequency of other congenital anomalies in patients with camptodactyly. Apparently, these associated anomalies are more common in the congenital than in the acquired cases. Adams[1] found a high incidence of hammer toe in families with camptodactyly, both in affected and nonaffected members. Murphy[22] reported the combination of camptodactyly and subluxation of the knee joint in 5 generations. Maurer[19] found a high incidence of pectus excavatum, high arched palate, and other developmental anomalies. In our series of 19 patients, 10 had other congenital anomalies, and in 6 of these patients these anomalies were of a severe nature (Table 1).

ROENTGEN FINDINGS

The affected finger is best studied roentgenographically in the true lateral projection. The findings in our patients of whom lateral roentgenograms of the affected little fingers were available are shown in Figure 6, *A–D* and Figure 7, *A–E* (see Figure 8, *A–H* for comparison with normal little fingers). The skin of the flexor aspect is usually shortened to a varying degree. The proximal phalanx often tapers distally. The head of the first phalanx is commonly bent or tilted volarward; its surface is generally

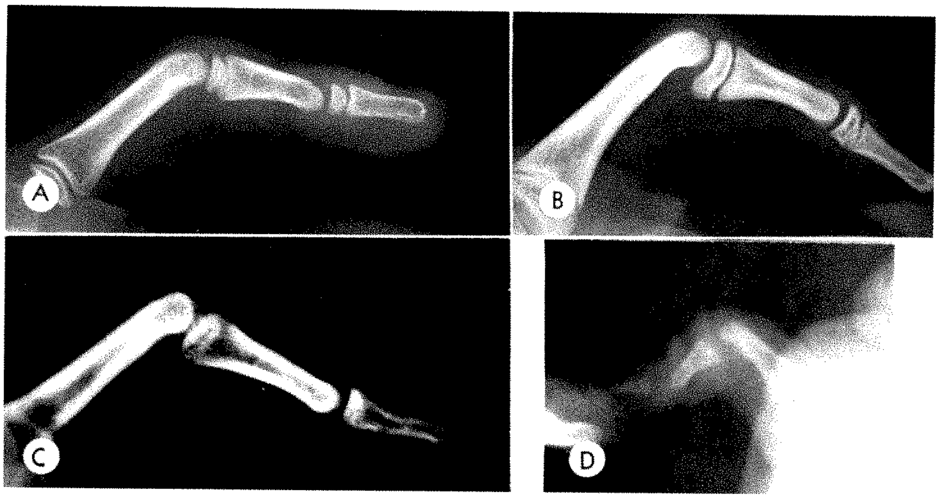

Fig. 6. (*A–D*) Lateral roentgenograms of the affected little finger. (*A*) Patient 1. (*B*) Patient 5. (*C*) Patient 10. (*D*) Patient 17.

smooth, but occasionally it is slightly flattened dorsally. A mild to moderate volar subluxation of the proximal end of the second phalanx is present in some cases. Sometimes the concavity or depression of the proximal articular surface of the second phalanx is located more dorsally than normally. The proximal part of the second phalanx commonly appears enlarged in its palmar aspect, but the finding may be due to rotation of the finger rather than to a true overgrowth of bone.

Essentially similar bone changes have been described by previous authors but usually on the basis of single cases.[5,12,13,18,19,23,29] The findings in the present series of patients suggest that although common, the roentgen changes in the bones are not constant. Furthermore, their presence or absence does not appear to have any clear relation to the duration or degree of severity of the disorder or other obvious factors.

PATHOLOGY AND PATHOGENESIS

A number of soft tissue and osteoarticular changes has been described in the affected fingers on the basis of observations made at operation and a few anatomic dissections of amputated fingers. The findings or combination of findings described have varied with the authors.

The soft tissue changes mentioned in the literature include retraction of the skin and subcutaneous tissue of the volar aspect of the affected finger, shortening of the flexor tendons, shortening of the tendon sheath, shortening of the ligaments, and shortening of the articular capsule. The already described longitudinal cord-like bands, which can be felt under the skin of the flexor side, have been defined by Adams[1] as thickened terminal cutaneous filaments of the fascia and by Neuhoff and Oppenheimer[24] as a combination of contracted skin, subcutaneous tissue, articular ligaments and capsule.

Of the osteoarticular changes, the most commonly mentioned are a volar inclination or rotation of the head of the first phalanx, and a volar subluxation of the proximal and the second phalanx.[19,23,26,29] According to some authors,[8,9,10,26] the head of the first phalanx is divided into a volar surface which articulates with the second phalanx, and a dorsal atrophic surface. Secheyron and Deforges-Mériel[30] have found that the proximal articular surface of the middle phalanx is divided by a ridge into a larger dorsal facette which articulates with the head of the first phalanx and a volar nonarticular surface; the latter is located on a bony prominence on which insert the shortened anterior and lateral ligaments.

The authors are divided as to which of

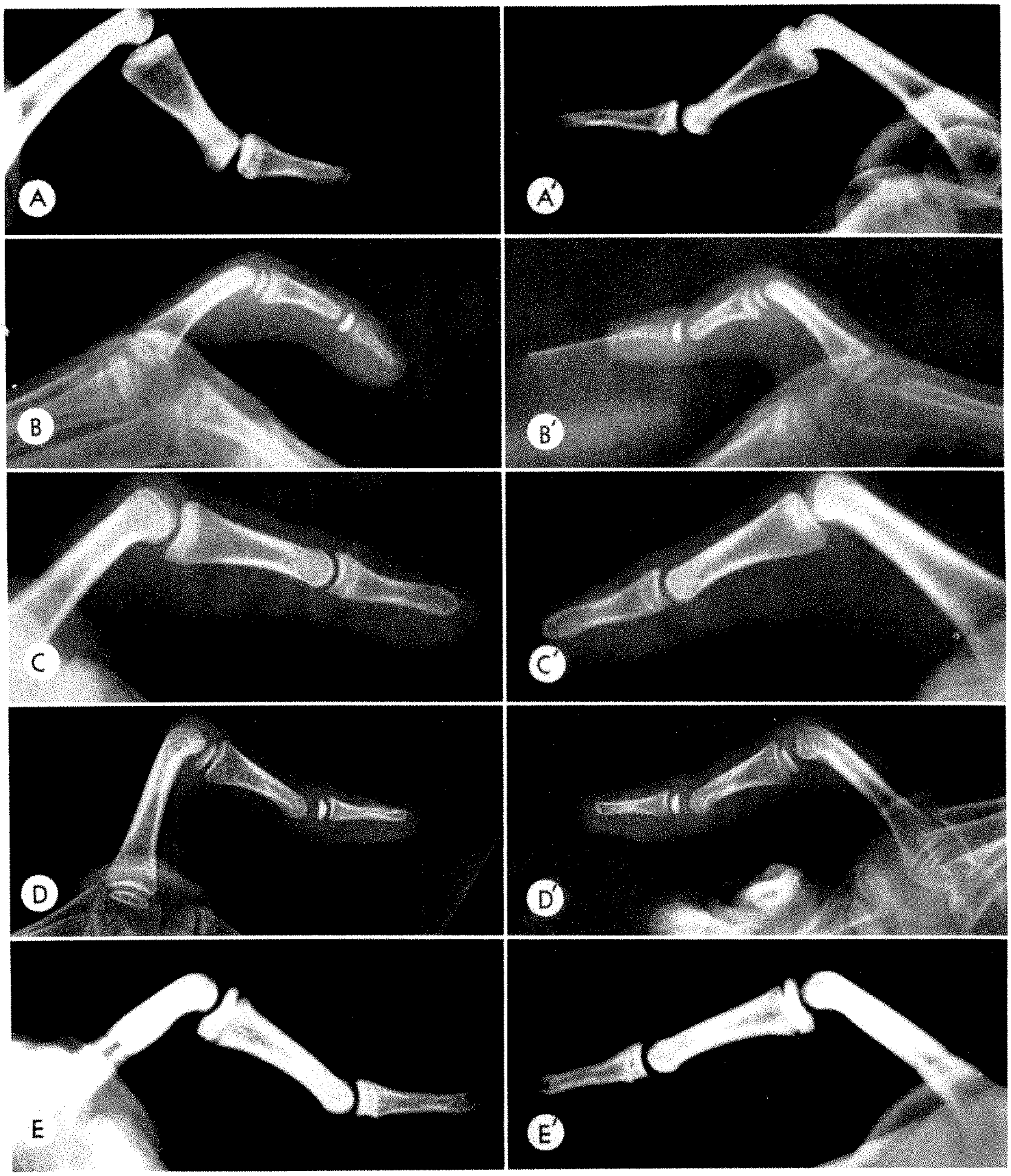

Fig. 7. (*A–E*) Lateral roentgenograms of both affected little fingers. (*A*, *A′*) Patient 4. (*B*, *B′*) Patient 8. (*C*, *C′*) Patient 13. (*D*, *D′*) Patient 14. (*E*, *E′*) Patient 18.

these anatomic changes is the primary abnormality responsible for the contracture. Some believe that the primary abnormality is one or more of the osteoarticular changes described above and that the soft tissue changes are secondary to long-standing flexion.[18,23,26,30] Others maintain that the primary lesion is in the soft tissues and that the bony changes are secondary phenomena. The latter view is the more widely held, but which of the soft tissues is primarily at fault is debated. Suggested possibilities have included: (1) an imperfect development and weakness of the extensor muscles (lumbricales and interossei) of the second and third phalanges;[1] and (2) a shortening of the flexor skin[15,33] of the lateral ligaments,[2] of the capsule,[34] of the articular ligaments plus capsule,[24] or of the flexor tendons.[12,13,19,32] The lack of agree-

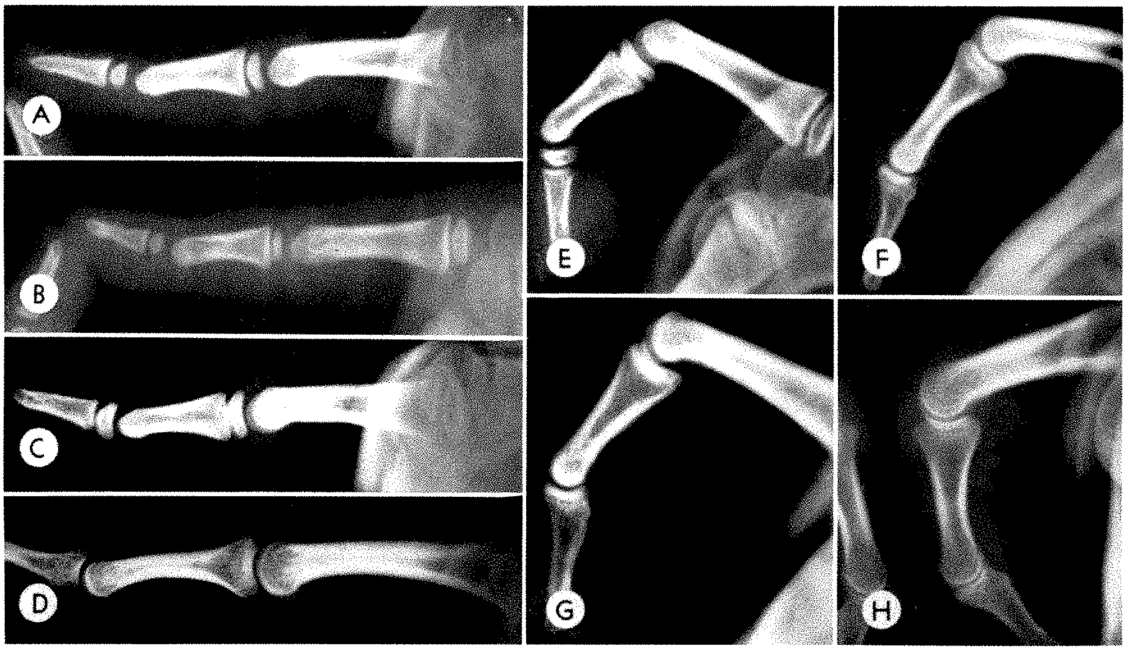

Fig. 8. (*A–H*) Lateral roentgenograms of a little finger of 8 normal individuals of various ages. Four fingers are in extension and 4 in flexion.

ment is largely due to the fact that all the anatomic changes described in the literature were observed in well developed or severe cases. It is obvious that the problem will not be settled conclusively until more is known about the changes which may be present early in the disease.

ETIOLOGY

The etiology of camptodactyly is entirely unknown. The congenital form is generally thought to be a developmental anomaly. Weber[35] suggested that the acquired form may be related to Dupuytren's contracture and is possibly due to repeated minor trauma, but this view is open to question.

DIFFERENTIAL DIAGNOSIS

Clinodactyly is a congenital and often hereditary anomaly of the little finger due to hypoplasia of the middle phalanx, mainly in its medial half; the involved finger is bent with concavity toward the radius, but is not flexed. In *arthrogryposis multiplex congenita* the fingers are often flexed, but other joints are also regularly involved, either in flexion or extension. The congenital deformity of the hands and fingers of *trisomy 17–18* is characteristic: the hands are held in moderate ulnar deviation, all the fingers are in marked flexion contracture, and the thumb, index and little fingers overlap the middle and ring fingers. The so-called *deviation of the fingers "en coup de vent"* is a rare congenital deformity of the hands apparently secondary to a developmental defect of the palmar fascia; it is characterized by deviation of all the fingers toward the ulna. Fexion of the fingers occurs also in a variety of *severe congenital deformities of the hands*, such as clubbed hands, lobster claw hands, syndactyly, polydactyly, brachydactyly, symphalangism, and hypo- and hyper-phalangism; in some of these cases the finger contracture is similar in appearance to camptodactyly and may have the same pathologic origin. *Trigger finger* (locking or snapping finger) is an affection in which the movement of one finger is usually arrested temporarily in flexion or extension and then continues with a jerk. The condition is most often acquired in adult life and results from a localized stenosing tenovaginitis. In children it is rare and is usually limited to the thumb. A rare congenital form also limited to the thumb is mentioned in the literature. *Advanced rheumatoid arthritis* may likewise give rise to a flexion deformity of the fingers, but in this disorder the flexion contracture characteristically predominates at the metacarpophalangeal joints, and other joints in the body are also affected. Contracture of fingers may occur in a large number of neurologic disorders of peripheral or central origin, or may follow *infection*, *burns*, or *trauma*. *Volkmann's ischemic* contracture of the fingers is a condition characterized by degeneration, atrophy and contracture of muscles, resulting from prolonged interference with the normal circulation of the forearm and hand by tight bandage, splinting, cast, hemorrhage from fracture, injury to a large artery, and possibly from exposure to cold. "*Bilateral incurving of the distal phalanx of the little finger*" is a rare disorder characterized by a progressive palmar and radial deviation of the distal phalanx of the little fingers and by an increase in depth of its epiphyseal line. It develops apparently between the eighth and fourteenth year of life. Its etiology is unknown. *Dupuytren's contracture* simulates camptodactyly more closely than any other condition. However, in this disease the finger contracture is due to a thickening, hardening and shortening of the palmar fascia, which is normal in camptodactyly. As opposed to camptodactyly, the skin of the palmar aspect of the hand and fingers in Dupuytren's contracture is abnormal, showing ridges, nodules and puckerings. The disorder has its onset in middle age or in older adults, is seen chiefly in males, and is continually progressive. The flexion is characteristically at the metacarpal phalangeal joint and is generally greater than 90°. The ring finger is more often affected than the little finger.

TREATMENT

In most instances camptodactyly is of little clinical significance and requires no therapy or only conservative therapy. The conservative measures advocated (particularly for the first stage of the disorder) have included prolonged splinting of the affected fingers in extension, active exercises, gentle passive stretching, and massages. Surgical procedures may have to be considered in severe cases to improve the function or the appearance of the hand. The surgical measures mentioned in the literature have varied with the opinions of the authors as to what constitutes the anatomic lesion or lesions responsible for the contracture and have included the following, employed singly or in combination: plastic procedures designed to lengthen skin of the flexor aspect of the affected finger; section of all the subcutaneous bands; section and/or lengthening of the flexor tendons; section of the articular ligaments; capsulotomy; wedge osteotomy in the neck of the first phalanx in order to shorten and straighten this bone; resection of the head of the fifth phalanx; resection of the entire distal part of the first phalanx; and resection of the entire articulation. A prolonged after-treatment with extension splinting is generally recommended in all these procedures. Amputation has also been mentioned as a necessary form of treatment in exceptional instances.

None of the conservative or surgical forms of therapy so far devised has produced consistently satisfactory results either in halting or remedying the contracture. Each method has been followed by a considerable percentage of failures. In our series, 4 patients were treated conservatively without improvement; and 4 were treated surgically: 2 without improvement, and in the other 2 the condition was made worse by the operation.

SUMMARY

A review of the main features of camptodactyly, a special form of flexion contracture of the fingers, is presented on the basis of previous reports and 19 new cases.

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ARTHROGRAPHY AND ARTERIOGRAPHY IN PIGMENTED VILLONODULAR SYNOVITIS OF THE KNEE*

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PIGMENTED villonodular synovitis is a benign inflammatory condition characterized by a proliferation of the synovial lining of a joint, bursa, or tendon sheath.[7,8] Although approximately 200 cases of articular involvement have been reported,[17] roentgenographic observations have mainly dealt with soft tissue and local osseous changes; special roentgenologic procedures have rarely been utilized in diagnosis. To our knowledge, positive arthrograms have been reported in only 3 cases[2,3,9] and brief mention is made of the arteriographic findings in this condition.[10] The purpose of this paper is to (a) describe the positive arthrographic and arteriographic results in a case of pigmented villonodular synovitis of the knee, (b) point out the excellent roentgenologic-pathologic correlation, and (c) emphasize the similarity of the angiographic pattern to that seen in malignancy and the consequent undependability of arteriography in lesions of this nature.

REPORT OF A CASE

A 31 year old white male was admitted with a 4 year history of spontaneous effusions of the right knee joint. Physical examination disclosed a markedly enlarged right knee with a "boggy" synovium and obvious effusion extending approximately 6 inches above the superior aspect of the patella.

Routine laboratory work was within normal limits. One hundred and twenty cubic centimeters of clear yellow fluid were aspirated from the knee and an analysis of the synovial fluid disclosed a uric acid of 7.2 mg. per cent, a cholesterol of 205 mg. per cent, and no white blood cells.

Routine roentgenograms of the knee revealed a narrowed joint, normal juxta-articular bones, and a large soft tissue swelling in the suprapatellar area (Fig. 1, *A* and *B*). Femoral arteriograms demonstrated a very vascular mass involving the suprapatellar area with irregular vessels and a diffuse contrast "stain" (Fig. 2, *A*, *B* and *C*). Arthrograms, using 50 per cent hypaque,* showed a markedly enlarged, distorted suprapatellar bursa composed of numerous recesses and filling defects (Fig. 3, *A*, *B* and *C*).

Radical synovectomy of the right knee was accomplished with removal of 95 per cent of the synovium. Following surgery, radiation therapy was instituted and 3,000 r was delivered over a period of 4 weeks to the operative site.

The pathologic specimen was encapsulated and measured 12.5×14.5×4 cm. The center of the mass appeared brown in color and presented numerous villous projections. Sections revealed many papillary folds, and the stroma contained an abundance of hemosiderin-laden histiocytes. In addition, there was moderate infiltration of lymphocytes and plasma cells. Occasional multinucleated giant cells were seen while numerous foam histiocytes were also evident. The surrounding tissue was fibrovascular in nature and revealed a mild chronic inflammatory infiltration.

DISCUSSION

First described in 1865 as a circumscribed articular lesion,[16] in later reports similar cases appeared under a variety of names such as xanthoma, xanthogranuloma, giant cell tumor, chronic hemorrhagic villous synovitis, giant cell fibrohemangioma, fibrohemosideric sarcoma, sarcoma fusi-

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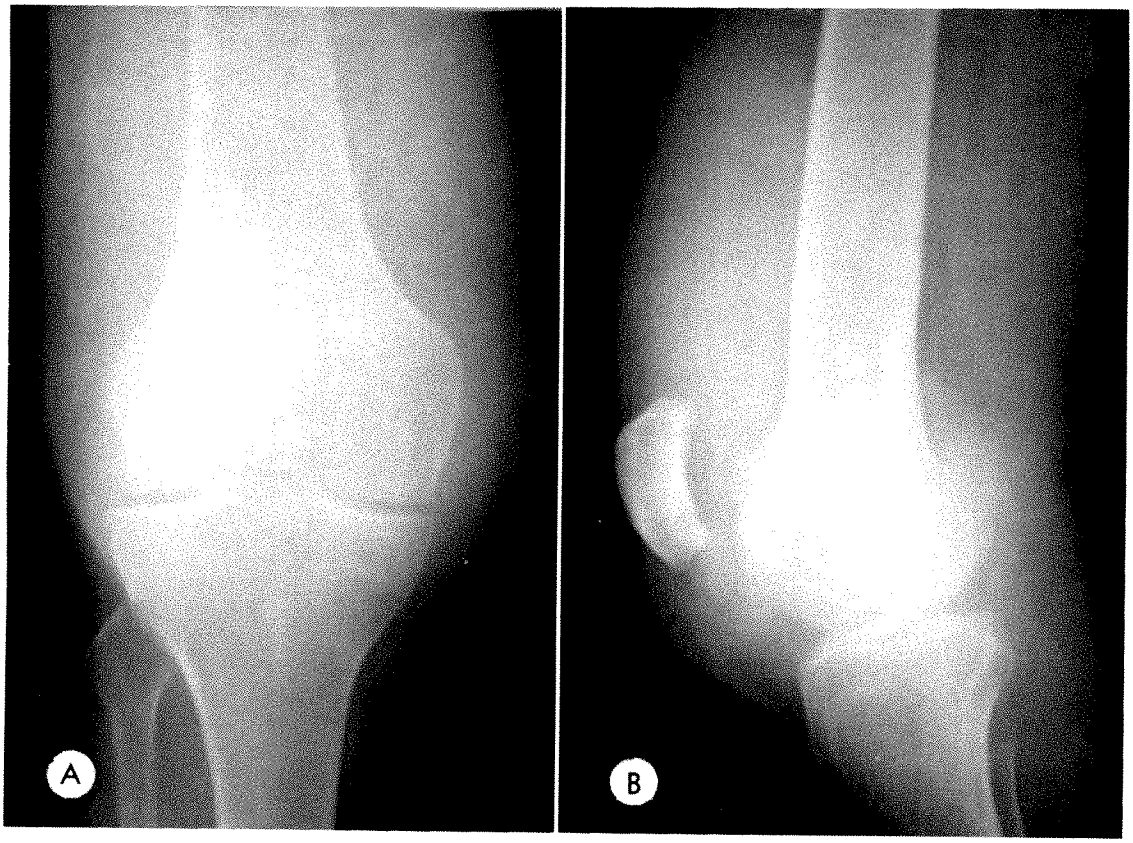

Fig. 1. (*A* and *B*) Marked expansion of suprapatellar and infrapatellar bursae by fluid and synovial tumefaction. Joint space is narrow and no bone erosions are noted.

giganto-cellulare, benign polymorphocellular tumor of synovial membrane, and malignant polymorphocellular tumor of synovial membrane.[8,17] After reviewing the literature and their own cases, Jaffe *et al.*[8] recognized common gross and histopathologic characteristics and designated the condition as "pigmented villonodular synovitis, bursitis or tenosynovitis" depending on the site of involvement.

Pathologically, the lesion may be either diffuse or nodular—involving the entire synovial cavity or affecting a limited area in an otherwise normal synovium. The gross appearance is characterized by the formation of brownish pigmented villous and nodular outgrowths which, following fusion and intermingling, may resemble a complex spongy pad. Microscopically, an intergrowth and fusion of nodular and villous proliferations covered by synovial lining cells are noted. There are large numbers of polyhedral and spindle-shaped stromal cells while multinucleated giant cells and histiocytes, some loaded with cholesterol, are also found. Hemosiderin is found both intra- and extracellularly, and thin walled vascular channels are scattered throughout the tissue of the lesion.[1,4,7,17]

Pigmented villonodular synovitis is a disease of young adults. Age, at time of diagnosis is most frequently in the 3rd and 4th decades. The knee is most frequently involved with lesser sites of involvement being the ankle, hip, tarsus, and carpus followed by the elbow and shoulder. Swelling (except in the hip) and minimal to moderate pain are the most common complaints and the usual initial symptoms. Limitation of motion and locking of the involved joint are also described. Tenderness with swelling or focal masses may be

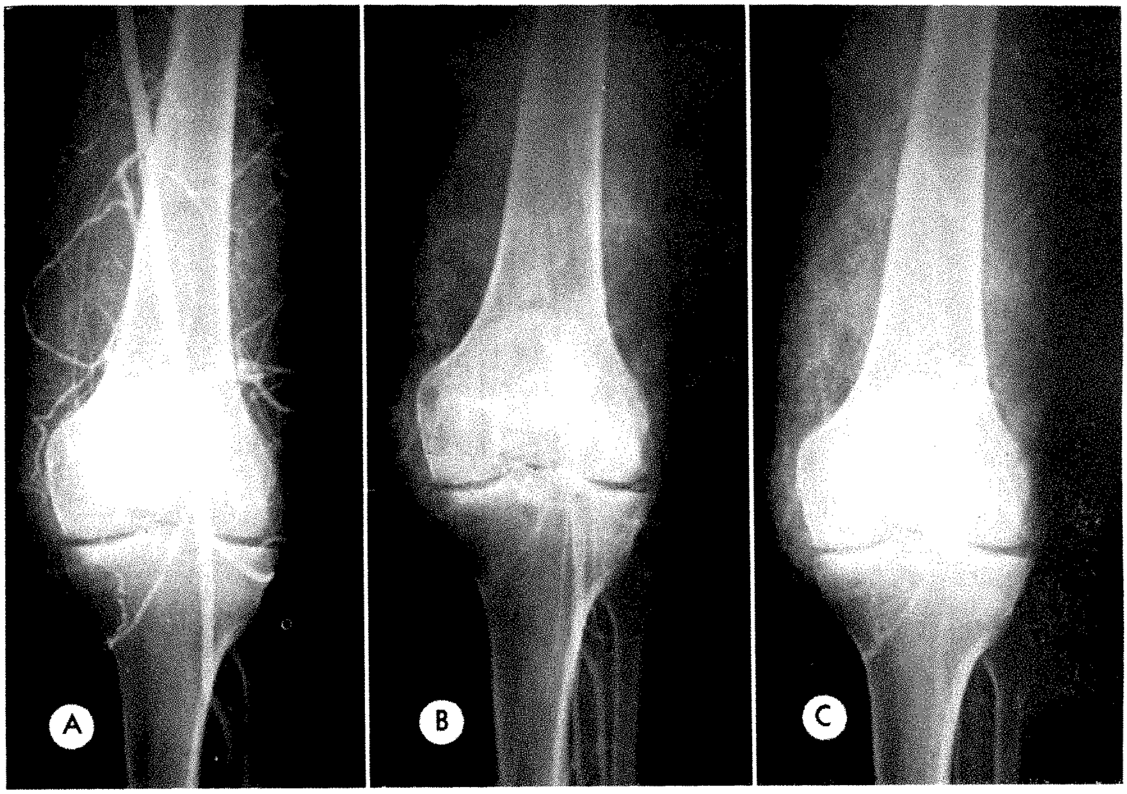

FIG. 2. Femoral arteriograms. (*A*) Early arterial phase. (*B*) Late arterial phase. (*C*) Capillary and early venous phase. Marked hypervascularity with no vessel displacement. Vessels of varying caliber are seen and diffuse contrast "stain" demonstrates extent of mass. Slight arteriovenous shunt is noted.

palpable about the joint. Typically, the clinical course is reported to be progressive with remissions and exacerbations. Effusions occur and aspiration usually yields xanthochromic or sanguineous fluid that may be high in cholesterol. Fever, leukocytosis and elevated erythrocyte sedimentation rate are usually not found.[1,7,17]

The roentgenographic appearance of this condition as it occurs in the knee is characterized by the presence of fluid in the joint and an excessive amount of synovial thickening, which may appear smooth and homogeneous or partly nodular in outline. An elevated iron content of the tissues may amplify the density of the synovitis. Joint spacing is symmetric and normal, and there is no evidence of osteoporosis. In addition, the disease is usually monarticular. Sharply circumscribed erosions having thin sclerotic margins have also been described in the bones adjacent to the involved joint. These osseous erosions are attributed to pressure by the soft tissue mass on articular cartilage and cortical bone. The scarcity of osseous lesions about the knee is explained by the large synovial space of the joint allowing expansion of the mass without undue pressure.[4,7,12,17]

Positive arthrographic findings have been reported in 3 cases—in 2 air was employed and 1 was performed with positive contrast material.[9,33] The roentgenographic findings are characterized by enlargement of the suprapatellar bursa with soft tissue lobulations projecting into the gas shadow. Pneumoarthrograms, however, have been described as negative in 4 additional cases.[17]

In the presented case the markedly enlarged, distorted suprapatellar bursa with

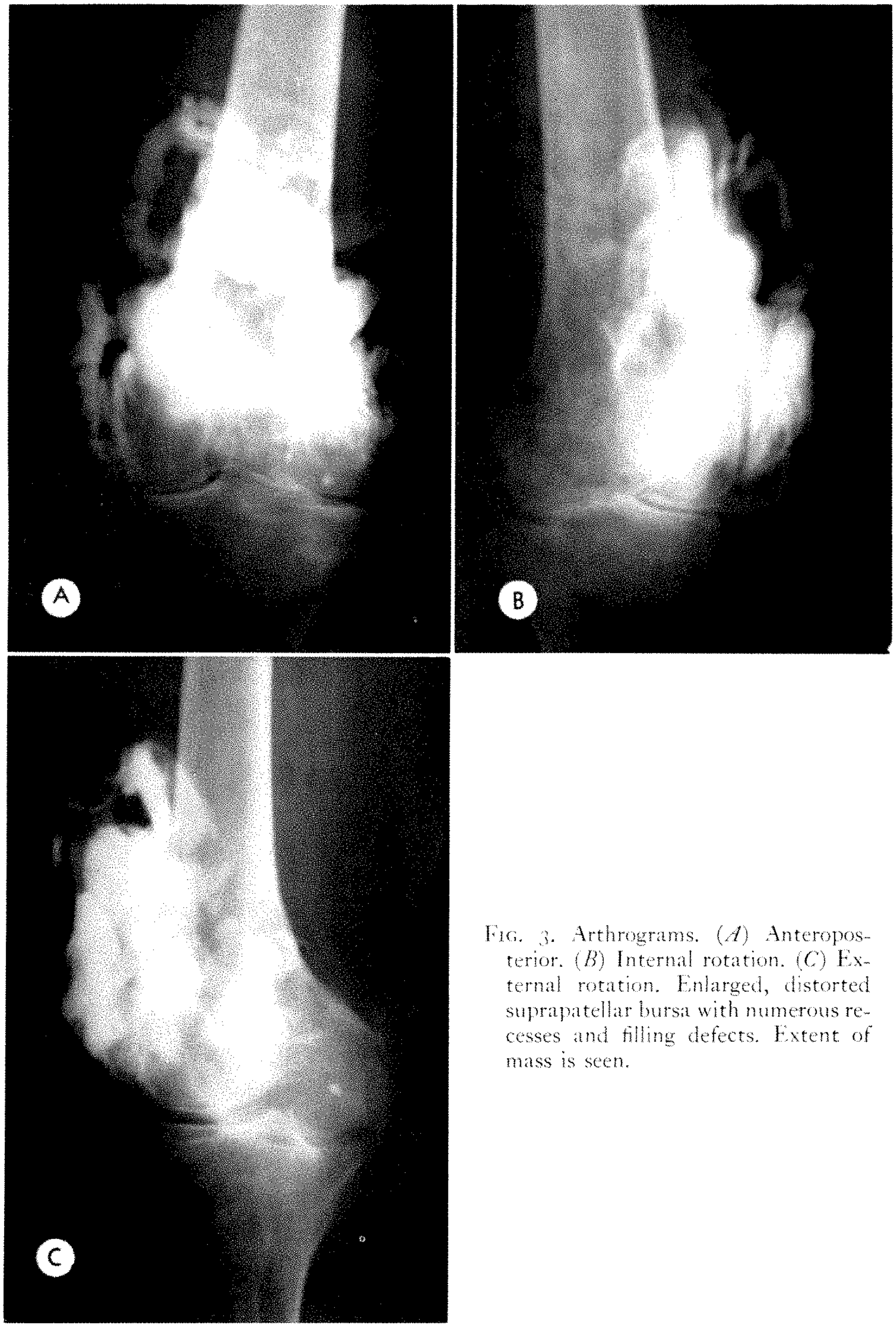

FIG. 3. Arthrograms. (*A*) Anteroposterior. (*B*) Internal rotation. (*C*) External rotation. Enlarged, distorted suprapatellar bursa with numerous recesses and filling defects. Extent of mass is seen.

numerous recesses and filling defects, demonstrated by arthrography utilizing positive contrast material, correlates well with the gross pathology. In addition, the authors are aware of similar unpublished findings in another case.[15] Since air is considered less satisfactory than positive contrast material in arthrography,[6] more experience with the latter may prove this procedure to be diagnostic in the great majority of cases.

Only brief mention is made of the arteriographic findings in a case of pigmented villonodular synovitis of the knee.[10] The appearance is characterized by a highly vascular mass with a slight arteriovenous shunt. The arteriogram in the presented case demonstrates well the extent and increased vascularity of the tumor. Diffuse "stain," small arteriovenous shunt, and irregular vessels are also seen.

The similarity of these findings to the angiographic pattern found in malignant tumors is noteworthy. Numerous authors[5,10,13,14,18,19,20] have described the arteriographic signs of malignancy in tumors of the extremities. These include: irregular arteries around the tumor periphery, presence of lacunae due to irregular collections of contrast material, arteriovenous shunts, abrupt termination of arteries, straight veins coursing at right angles to the level of venous flow, nests of small vessels encircling an avascular area, and hypervascularity with pathologic vessels, deployed seemingly without purpose or course. In benign lesions, however, one usually sees only mechanical displacement of vessels. Diffuse "staining" of the mass by contrast material is seen in both benign and malignant tumors.

Applying these criteria to the present case, one might erroneously advance a diagnosis of malignancy. Lagergren *et al.*,[11] however, point out that certain inflammatory processes can be highly vascularized, especially chronic processes with infiltration of round cells. They also state that synovial tissue may be hypervascularized and suggest that angiography should not be relied on for diagnosis of highly vascularized masses in the vicinity of tendon sheaths and joints.[10]

Probably the greatest value of arteriography in such cases is in the delineation of the extent of the tumor. Other information which may be derived is the amount of the vascular shunt within the tumor and its influence on the vascular status of the rest of the extremity.

SUMMARY

A case of pigmented villonodular synovitis of the knee is reported. Emphasis is placed on the positive arthrographic and arteriographic findings and the excellent roentgenographic-pathologic correlation. In addition, the similarity of the angiographic pattern to that found in malignant tumors is noted, and the consequent undependability of arteriography in lesions of this nature is stressed.

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POSTSTENOTIC DILATATION (ANEURYSM) OF THE SUBCLAVIAN ARTERY ASSOCIATED WITH CERVICAL RIB*

REPORT OF TWO CASES

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IN 1957, 2 cases were reported in which poststenotic dilatation (aneurysm) of the right subclavian artery was visualized by intravenous angiocardiography.[14] The percutaneous transfemoral selective arteriographic technique of visualization of the subclavian artery[15] was used to demonstrate the vascular dilatation in one of the cases herein reported. This method appears to be a more precise way of demonstrating the lesion and is the main reason for this report.

REPORT OF CASES

CASE I. A 19 year old seamstress was admitted because of intermittent attacks of pain in the neck and left shoulder with numbness and tingling of the arm and fingers of the left hand. A pulsatile nontender mass was palpated along the left supraclavicular region. Neurologic examination showed signs of left brachial plexus irritation. Roentgen examination of the thorax revealed a rudimentary left cervical rib (Fig. 1*A*). Percutaneous transfemoral selective left subclavian arteriography disclosed poststenotic dilatation of the left subclavian artery (Fig. 1*B*). Surgical exploration of the left supraclavicular area verified the diagnosis. Freeing of the subclavian artery and neighboring nerve structures after section of the anterior scalene muscle without removal of the cervical rib relieved the poststenotic dilatation and the patient's complaints were subsequently alleviated.

CASE II. A 47 year old housewife was admitted because of a pulsatile mass in the left supraclavicular region and weakness and tiredness of the left upper extremity after overexertion of the arm. The mass measured 2 by 2 cm. and did not exhibit a bruit. The blood pressure in the right arm was 110/70; in the left, it was 90/70 mm. Hg. Visualization of the left subclavian artery was recommended but declined due to the fact that the clinical features were considered sufficiently diagnostic since the artery was adjacent to a palpable left cervical rib (Fig. 2*A*). At operation on June 25, 1963, the left subclavian artery beyond the cervical rib was found to be dilated (Fig. 2*B*). The left anterior scalene muscle was divided and immediately the constriction of the subclavian artery was relieved. The cervical rib was not removed and the patient made an uneventful recovery. Subsequently, the left supraclavicular mass disappeared and the blood pressures in both arms were equal.

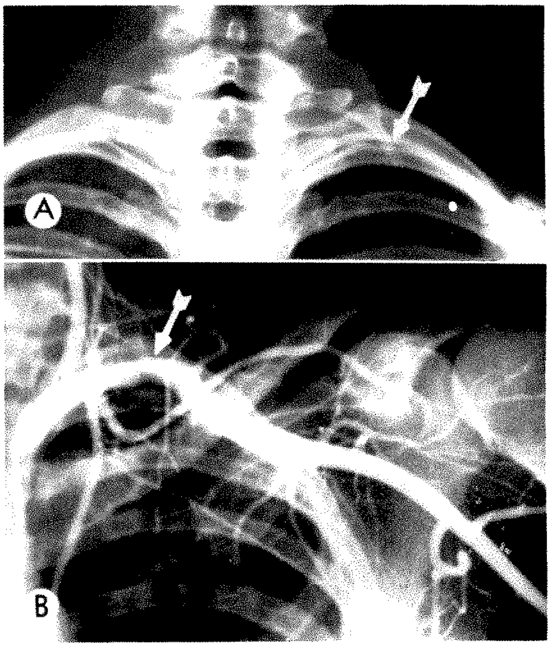

FIG. 1. (*A*) Roentgenogram showing the rudimentary left cervical rib (arrow). (*B*) Selective left subclavian arteriogram (courtesy of Dr. Mordecai Halpern) showing the poststenotically dilated left subclavian artery beyond the cervical rib (arrow).

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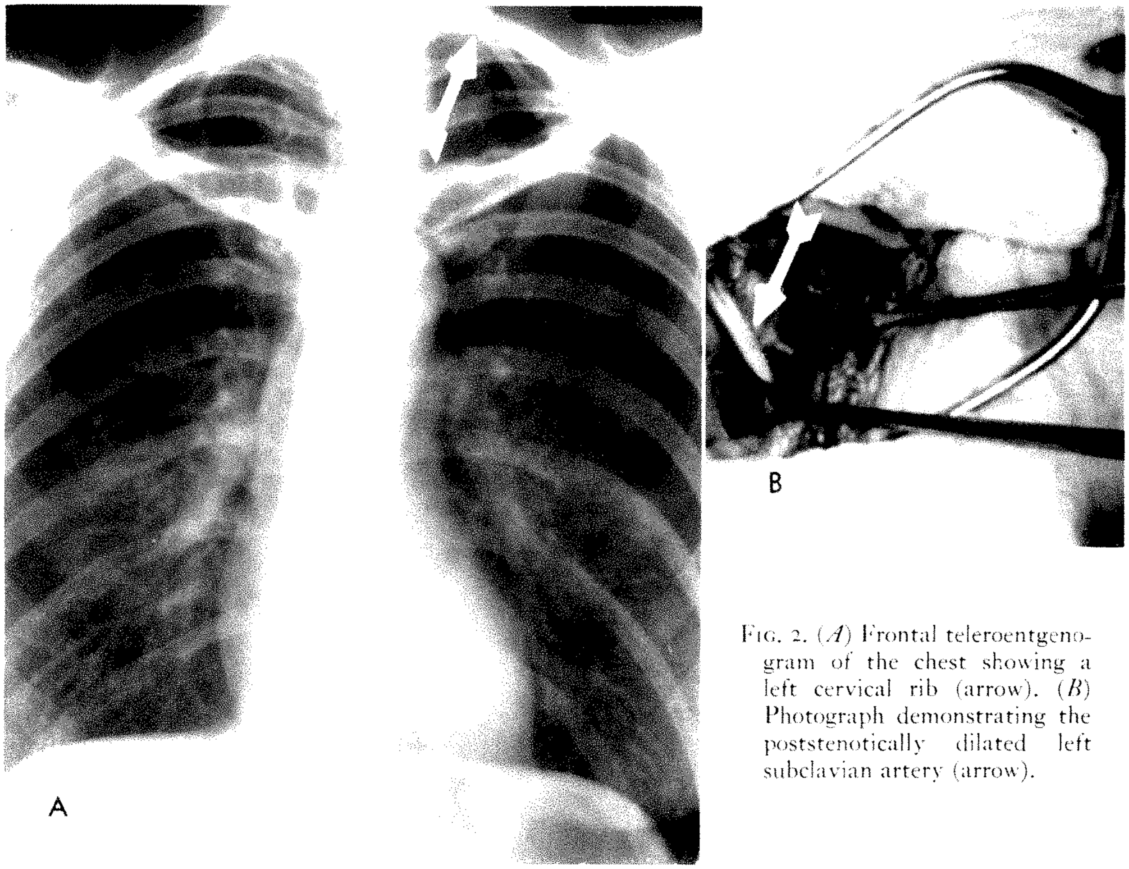

FIG. 2. (*A*) Frontal teleroentgenogram of the chest showing a left cervical rib (arrow). (*B*) Photograph demonstrating the poststenotically dilated left subclavian artery (arrow).

DISCUSSION

Poststenotic dilatation of the third part of the subclavian artery has long been recognized as a complication of a cervical rib. Indeed, Halsted[4] in 1916 was able to reproduce the lesion in the experimental animal and show that it followed physical and hemodynamic principles. Cervical ribs are not uncommon and have been reported in 0.5 to 0.7 per cent of routine chest roentgenograms.[16] Malformations of the first thoracic rib may also produce similar neurovascular compression.[3,16] The vast majority of patients with cervical or anomalous first thoracic ribs are asymptomatic and require no treatment. A few have brachial plexus neuritis due to impingement of nerves by incomplete cervical ribs.[2,9] A still smaller number, estimated by Adson[1] to be about 5.6 per cent of the patients with cervical ribs, present with vascular symptoms, chiefly Reynaud's phenomenon. Since subclavian artery thrombosis may occur even with mild neurovascular compression symptoms, surgical intervention for prophylactic as well as therapeutic reasons is advocated in every case.[11,13] There is general agreement that removal of a cervical or anomalous first thoracic rib and transection of the anterior scalene muscle are curative.[2,9,16]

Only about 70 cases of poststenotic dilatation (aneurysms) of the subclavian artery owing to a cervical rib have been reported in the literature. It may be that many of these cases are included among reports of the scalenus anticus syndrome and are not separated as instances of subclavian artery dilatation or aneurysm. Lindskog and Howes[6] in 1937 reported a case and, at that time, found only 29 cases in the concurrent literature. Other cases have since then been reported by Eden,[3] Mac Fee[7], Poppen and associates,[8] Imler *et al.*,[5] Ross,[10] and

Schmitz-Draeger.[12] Lindskog and Howes[6] emphasized the difficulty of making a diagnosis of subclavian artery aneurysm on clinical grounds alone, and advised verification by operation. However, this is no longer necessary since contrast visualization of the subclavian arteries can be achieved either with angiocardiography,[14] or with percutaneous transfemoral or axillary selective arteriography and the diagnosis can be established preoperatively.

SUMMARY AND CONCLUSIONS

Poststenotic dilatation (aneurysm) of the subclavian artery may follow compression by a cervical or anomalous first thoracic rib. Very often associated neurovascular symptoms may also occur, and these, even when mild, require surgical treatment, such as resection of the cervical rib and transection of the anterior scalene muscle in order to release the compression and to alleviate symptoms. These procedures will also prevent thrombosis of the involved artery. Preoperative verification of poststenotic dilatation is desirable and may be secured by angiography. Although by intravenous angiocardiography the involved area can be visualized, a more direct and distinct demonstration of the lesion may be obtained by percutaneous transfemoral or axillary selective arteriography of the involved subclavian artery.

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POSTERIOR CARDIAC PROMINENCE IN EBSTEIN'S ANOMALY SIMULATING MITRAL DISEASE*

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ALTHOUGH radiologic literature concerning Ebstein's anomaly has dealt primarily with angiocardiographic findings,[4,6,7,9] a few articles[1,2,10] have been devoted to evidence on plain roentgenograms of this malformation. Cardiomegaly, often very marked, a globular configuration with a narrow base, right atrial prominence and decreased pulmonary blood flow are cited as hallmarks of this condition. Some individuals with this anomaly present a prominence of the cardiac silhouette posteriorly which may displace the esophagus in a manner somewhat simulating an enlarged left atrium of mitral disease. Since this manifestation of Ebstein's anomaly is not widely appreciated, it is felt desirable to report 2 illustrative cases seen at the University of Kentucky Medical Center and discuss the mechanism of this finding.

ILLUSTRATIVE CASES

Case I. J.C.M., a 26 year old male college student, was admitted to the University of Kentucky Medical Center for cardiac evaluation. At the age of 6 years, the patient was noted to have a heart murmur and was advised to limit his activity. Growth and development progressed normally. There was no history of cyanosis, shortness of breath, paroxymal dyspnea, orthopnea or edema. Rejection for military service occurred on two occasions because of an enlarged heart. There was no past history suggestive of rheumatic fever.

On physical examination the patient was well developed, slightly obese and in no distress. A Grade III rough apical systolic murmur extended throughout systole. No diastolic murmurs were heard even with exercise. A third heart sound was audible over the left fourth and fifth intercostal spaces near the sternal border.

Routine laboratory studies were within normal limits. An electrocardiogram demonstrated first degree atrioventricular block and complete right bundle branch block.

Chest roentgenograms (Fig. 1, *A* and *B*) revealed an enlarged cardiac silhouette with a globular contour. There was prominence of the left middle cardiac segment in the frontal projection. The base was very narrow and the main pulmonary artery segment and aortic knob appeared small. A distinct bulge was noted along the posterior margin of the cardiac contour on the lateral roentgenogram. Barium swallow studies showed a mild impression on the anterior surface of the esophagus in the area of the posterior bulge. Cardiac catheterization and angiocardiograms (Fig. 2) demonstrated a markedly enlarged right atrium. The opaque medium was sufficiently diluted by the large quantity of blood in the right atrium that no opacification distal to this chamber was visible. No evidence of an atrial shunt was demonstrated angiocardiographically or on catheterization data. The findings were interpreted as characteristic of Ebstein's anomaly.

Case II. M.G., a 48 year old white female, was referred to the University of Kentucky Medical Center for cardiac evaluation. The patient believed she had had rheumatic fever at the age of 6 years. At 24 years of age, a chest roentgenogram was said to reveal cardiac enlargement. She had observed intermittent cyanosis of the fingers, hands, and toes since her early teens. There had been a gradual onset of dyspnea on exertion at this time which slowly progressed over the succeeding years. At the time of the admission she was virtually incapacitated because of weakness and exertional dyspnea.

Physical examination revealed a well developed, well nourished individual in no distress. Obvious cyanosis of the hands, feet and lips was present. Mild pitting edema of the ankles was observed. A Grade IV rough, low-pitched presystolic murmur was best heard near the left sternal border in the fifth intercostal space. A low-pitched mid-diastolic murmur was audible in the same area.

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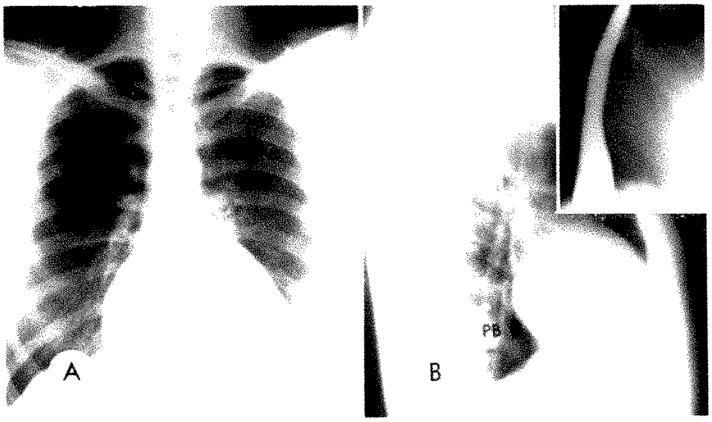

FIG. 1. Case 1. (*A* and *B*) Posteroanterior and lateral roentgenograms of the chest. (*Insert*) Right anterior oblique spot roentgenogram after a barium swallow. Note the narrow base, prominence of the left middle cardiac segment, posterior bulge (PB) and mild impression on the esophagus.

An electrocardiogram showed evidence of right atrial enlargement, first degree atrioventricular block and complete right bundle branch block. Roentgenograms of the chest (Fig. 3, *A*, *B* and *C*) demonstrated cardiomegaly with a globular contour and enlargement of the right side of the heart. The base was only slightly narrowed and the left middle cardiac segment was distinctly prominent. The lateral roentgenogram showed a definite bulge posteriorly which caused no appreciable impression on the barium-filled esophagus. Cardiac catheterization revealed a bidirectional shunt at the atrial level. Angiocardiograms showed a hugely enlarged right atrium and leftward displacement of the tricuspid valve typical of Ebstein's anomaly (Fig. 4, *A*–*D*).

MECHANISM OF POSTERIOR ENLARGEMENT OF THE CARDIAC SILHOUETTE IN EBSTEIN'S ANOMALY

Ebstein's anomaly is characterized by malformation, narrowing and downward displacement of the tricuspid valve. The valvular displacement significantly reduces the size of the functioning right ventricle with the portion of this chamber lying proximal to the displaced tricuspid valve being incorporated into and greatly enlarging the right atrium. Although infants with this malformation may show a relatively normal musculature in the atrialized portion of the right ventricle, thinning and bulging of this portion of the right atrium are a constant finding in patients who survive for any length of time. This suggests that bulging and atrophy of the musculature represent changes due to altered hemodynamics rather than a primary muscular disturbance with the valvular deformity and displacement. The ventricular wall distal to the tricuspid valve retains a normal right ventricular thickness.

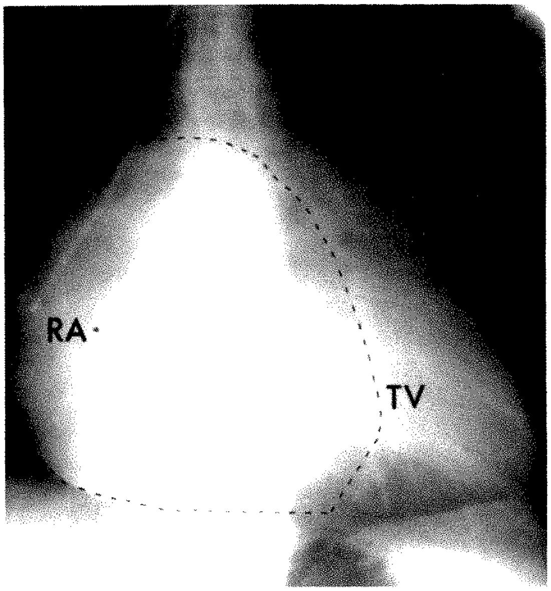

FIG. 2. Case 1. Anteroposterior angiocardiogram following injection of 60 cc. of opaque medium into the right atrium. The dotted line outlines the right atrium and the atrialized portion of the right ventricle. The right atrium (RA) is very large and the tricuspid valve (TV) is displaced far to the left.

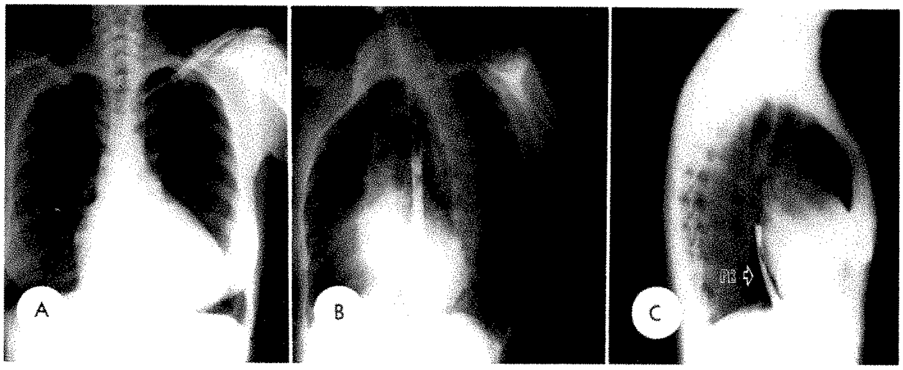

Fig. 3. Case II. (*A*, *B* and *C*) Posteroanterior, lateral anterior oblique and lateral roentgenograms of the chest following barium swallow. The posterior bulge (PB) is definite and the left middle cardiac segment is prominent.

The type of malformation of the tricuspid valve leaflets is variable. Fusion often is present. At times there is so much deformity it is difficult to identify individual leaflets. Upon opening the enlarged right atrial chamber a normally placed tricuspid annulus fibrosus usually can be distinguished as a distinct ridge dividing the true atrium from the atrialized portion of the right ventricle. The atrialized portion of the right ventricle, representing most or all of the normal inflow tract of this chamber, lies somewhat to the right of and posterior to the outflow tract. Consequently, enlargement of the atrialized right ventricle distal to the annulus fibrosis and proximal to the tricuspid valve proceeds in a posterior and right lateral direction. Bauer,[2] in an autopsy study of a patient with Ebstein's anomaly, placed barium in the right side of the heart and obtained roentgenograms. He was able to demonstrate the atrialized portion of the right ventricle as an identifiable separate abnormal segment of the right atrium located on the posterior and inferior portion of the cardiac contour. It is to be expected that, as dilatation of the abnormal right atrium increases posteriorly, a distinct bulge will appear on the posterior aspect of the cardiac contour and be visible on lateral roentgenograms. Before massive dilatation occurs, the posterior prominence represents the atrialized portion of the right ventricle. The posterior bulge may effect an impression on the esophagus, usually at a level slightly lower than the impression created by the enlarged left atrium of mitral stenosis, although this is not invariable. At times, it is impossible to differentiate the appearance created by an enlarged left atrium of mitral disease, particularly insufficiency, from the posterior bulge associated with Ebstein's anomaly.

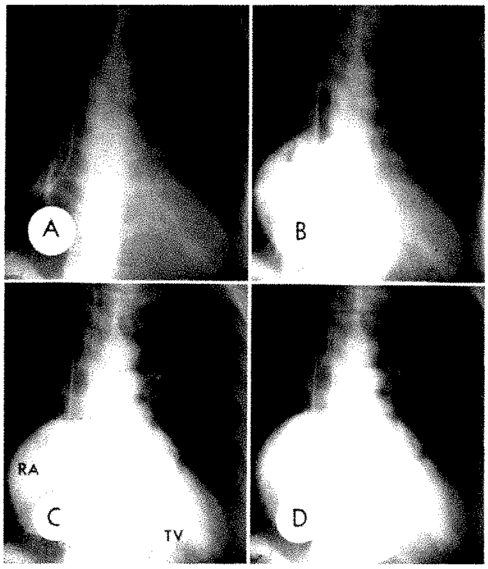

Fig. 4. Case II. (*A*–*D*) Anteroposterior angiocardiograms after injection of 50 cc. of opaque medium through a catheter with its tip in the right atrium. The dotted line outlines the right atrium and the atrialized portion of the right ventricle. The right atrium (RA) is markedly enlarged and the tricuspid valve (TV) is displaced far to the left.

With further dilatation the tricuspid annulus eventually becomes totally effaced and no distinct dividing line between the two portions of the right atrium is visible. When this occurs, the entire posterior cardiac border appears on the lateral roentgenogram as a smoothly contoured prominence with no suggestion of localization inferiorly. This posterior shadow results from dilatation of both the true right atrium and the atrialized portion of the right ventricle.

As is well known, a similar chain of events occurs with mitral disease and left atrial enlargement. Before the left atrium is massively enlarged, particularly in mitral stenosis, this chamber can be appreciated as a posterior prominence creating a localized impression on the barium-filled esophagus usually at a slightly higher position than the bulge seen in Ebstein's anomaly. With further dilatation, especially when associated with left ventricular enlargement as occurs in mitral insufficiency, localization of the bulge disappears and the entire posterior aspect of the cardiac silhouette is outlined by a much longer smoothly contoured prominence usually displacing the esophagus. In this instance the posterior bulge is the result of both left atrial and left ventricular enlargement.

DIFFERENTIATION OF EBSTEIN'S ANOMALY AND MITRAL VALVULAR DISEASE

Confusion between Ebstein's anomaly and mitral disease, particularly insufficiency, will not occur if characteristic clinical and roentgenographic manifestations are present. The clinical and electrocardiographic findings of Ebstein's anomaly, particularly if the patient is an adult, are quite distinctive. Excellent recent reviews[5,10] have dealt with the clinical findings of Ebstein's anomaly. About 85 per cent of these patients show some degree of cyanosis. Palpitations and bouts of paroxysmal tachycardia are very common. The murmurs heard, usually rough and systolic, tend to be maximal at a location more medial than the murmurs of mitral disease. A striking gallop rhythm is usually present in adults. The electrocardiogram characteristically shows complete right bundle branch block. Sudden death is not uncommon.

At other times clinical differentiation is more difficult. Approximately 15 per cent of these patients are not cyanotic and there may be few or no symptoms. In some patients, as in Case 1, the clinical differentiation of mitral disease and Ebstein's anomaly requires fine discrimination and hinges on rather subtle points. Since Ebstein's malformation is relatively rare, confusion between the two conditions is understandable in institutions seeing a limited number of patients with congenital heart disease. There is a report[3] in the literature of a patient with Ebstein's anomaly being surgically explored with a mistaken diagnosis of operably correctable mitral disease. At exploration a soft bulging prominence was felt posteriorly which eventually proved at autopsy to represent the atrialized portion of the right ventricle.

In typical cases little difficulty is encountered in the roentgenologic differentiation of these two entities. The narrow heart base, marked cardiomegaly with a globular contour, right atrial enlargement, sparse pulmonary vasculature and absence of valvular calcification all point toward Ebstein's anomaly rather than rheumatic disease. An enlarged left atrium of mitral disease frequently presents as a double contour on the posteroanterior roentgenogram and may elevate the left main stem bronchus. An enlarged left atrium more consistently causes an impression on the barium-filled esophagus than does the abnormal right atrium of Ebstein's anomaly.

There are other cases, however, in which differentiation on plain roentgenograms is much more difficult and, indeed, may be impossible. Difficulty is particularly likely to occur when the heart is not massively dilated. In such situations it may be impossible to determine whether the posterior bulge seen on the lateral roentgenogram represents an enlarged left atrium of mitral disease or the atrialized portion of the right atrium of Ebstein's anomaly. Although the localized posterior bulge of Ebstein's anom-

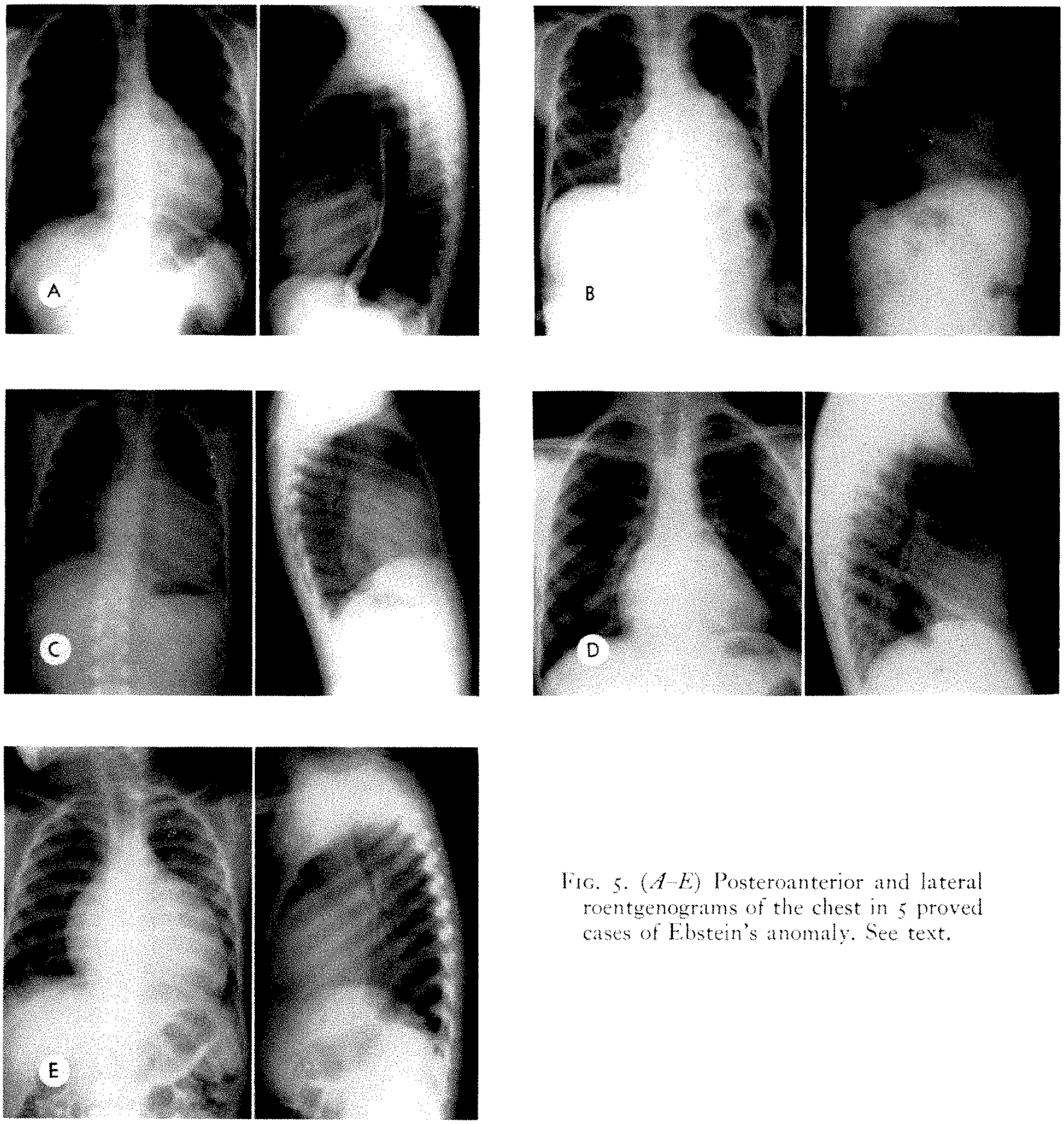

Fig. 5. (*A–E*) Posteroanterior and lateral roentgenograms of the chest in 5 proved cases of Ebstein's anomaly. See text.

aly is typically somewhat lower than that seen with left atrial enlargement, differentiation on these grounds is not always possible. Patients with Ebstein's anomaly may show a relatively normal pulmonary vasculature, although the typical patient will have pulmonary oligemia. There may be no globular contour, massive cardiomegaly or a narrow base. There is usually prominence of the left middle cardiac segment in both conditions, and approximation of the anterior surface of the heart to the dorsum of the sternum is of no differential value. When cardiomegaly is profound, the differential points between the two diseases cited above are usually evident, facilitating arrival at the correct diagnosis. Angiocardiography, of course, provides decisive differential information.

COMMENT

Although there are excellent recent reports[1,10] of the roentgenographic findings in Ebstein's anomaly, posterior prominence of

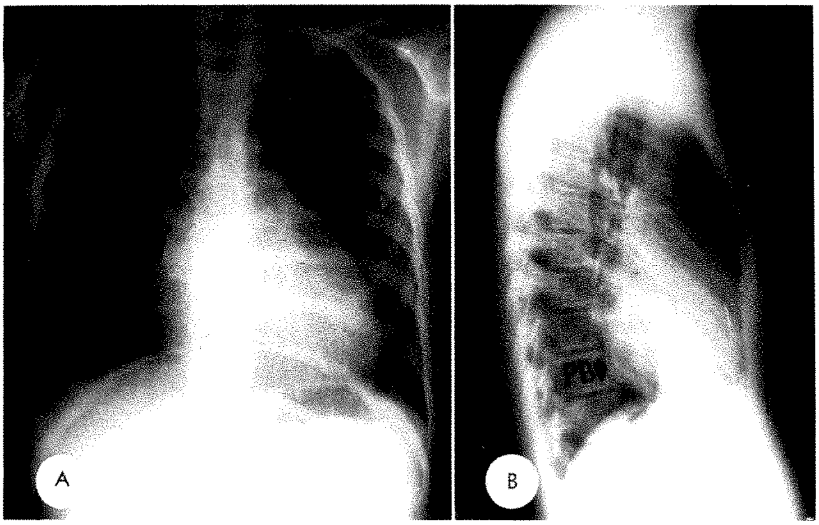

Fig. 6. (*A* and *B*) Posteroanterior and lateral roentgenograms of the chest of an 11 year old female with proved mitral insufficiency. The posterior bulge (PB) on lateral roentgenogram is located in a position virtually identical with that seen in Ebstein's anomaly.

the cardiac shadow as seen on the lateral roentgenogram is not emphasized. Very few lateral roentgenograms are included in these reports, although McCredie *et al.*[8] clearly illustrated posteroinferior bulging of the cardiac contour. Roentgenograms of patients with proved Ebstein's anomaly, obtained through the courtesy of a colleague (Fig. 5, *A–E*),* clearly show that posterior prominence of the cardiac silhouette is not uncommon in this entity. In contrast to the roentgenograms in 5*B*, those in 5*A* show decreased pulmonary vasculature suggesting Ebstein's anomaly. In both instances the posterior bulge was rather high and the left middle cardiac segment was prominent—findings not inconsistent with a diagnosis of mitral disease. On the other hand, the roentgenograms in 5*C* clearly show the posterior bulge to be placed inferiorly. In 5*D* the posterior prominence is more generalized, and the pulmonary vasculature and aortic shadow are not particularly remarkable. Cardiomegaly is marked in 5*E*, and the pulmonary vasculature is sparse. In this instance the posterior bulge is generalized, indicating enlargement of both the true right atrium as well as the atrialized portion of the right ventricle. All of these roentgenograms show prominence of the left middle cardiac segment. The lateral projections give more evidence of right-sided enlargement than was present in the cases reported above. Roentgenograms of an 11 year old girl with proved mitral insufficiency (Fig. 6, *A* and *B*) are shown for comparison.

Without clinical data, differentiation of Ebstein's anomaly and mitral valvular disease may be impossible, and even with adequate clinical evaluation, angiocardiography and cardiac catheterization may be required. If it is appreciated that the cardiac silhouette may show a localized posterior prominence in Ebstein's anomaly, confusion may be avoided. Careful consideration of all of the clinical, electrocardiographic and roentgenologic data usually permits an accurate differentiation.

SUMMARY

A posterior bulge on the lateral roentgenogram of patients with Ebstein's anomaly due to enlargement of the abnormal atrialized portion of the right ventricle is

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seen with considerable frequency. This finding has not been emphasized in the medical literature. Two cases demonstrating this appearance are reported. The probable mechanism of the posterior prominence is presented. The possibility of confusing this appearance with the enlarged left atrium of mitral valvular disease, as well as the differentiation on plain roentgenograms of the two entities, is discussed.

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EBSTEIN'S ANOMALY OF THE TRICUSPID VALVE*

ANGIOCARDIOGRAPHIC CONSIDERATIONS

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EBSTEIN'S anomaly of the tricuspid valve is an uncommon congenital cardiac malformation which often permits survival to adult life. It was first described by Wilhelm Ebstein[4] in 1866. In 1950, when Engle *et al.*[7] reviewed this anomaly, only 26 cases had been reported and none of these had been diagnosed prior to necropsy. In 1951, Soloff *et al.*[16,17] reported angiocardiographic visualization of the abnormal tricuspid leaflets in the first case conclusively diagnosed during life. Since then roentgenography and angiocardiography have become increasingly important in the diagnosis of Ebstein's anomaly. Our purpose is to present the angiocardiographic features of this anomaly in 7 selected patients demonstrating the wide range in tricuspid valve deformity and in the secondary functional tricuspid insufficiency which may be encountered.

ANATOMIC AND FUNCTIONAL CONSIDERATIONS

In Ebstein's anomaly the deformed tricuspid valve leaflets extend basket-like into the cavity of the right ventricle (Fig. 1, *A–E*). Fusion of the valve apparatus with the ventricular endocardium gives the malformed leaflets effective origin from the right ventricular wall. The anterior leaflet of the tricuspid valve is usually least affected.

Since the origin of the tricuspid valve is displaced into the right ventricular cavity, the proximal portion of the right ventricle is functionally incorporated into the right atrium. The muscular wall of this "atrialized" proximal right ventricle is atrophied[5] and thin, while the ventricular cavity, distal to the displaced tricuspid valve leaflets, is secondarily deformed and typically small. The right atrium is large and hypertrophied. Its effective size is further augmented by the asynchronously contracting "atrialized" proximal portion of the right ventricle, a region which functions as an atrial aneurysm. The left atrium and left ventricle are normal in size, but small relative to the right atrium.

Some elevation of right atrial pressure often occurs in Ebstein's anomaly. Frequently, this causes a right to left shunt of blood by way of a patent foramen ovale or less commonly an atrial septal defect. This shunt diminishes the stroke output requirement for the right ventricle compared to that of the left ventricle. In nearly half the cases, the shunt is sufficient to produce clinical cyanosis. The elevated right atrial pressure is most commonly the result of decreased compliance (or increased resistance to diastolic filling) of the malformed right ventricle. Not infrequently, a small diastolic pressure gradient across the deformed tricuspid valve or a small systolic pressure gradient in the pulmonary outflow tract can be detected.

At birth, the relatively high pressures normally found in the pulmonary artery together with restricted function of the right ventricle in Ebstein's anomaly result in abnormally high right atrial pressures. Then typically, cyanosis is observed due to right to left interatrial shunting via a patent foramen ovale. Rarely, an infant will succumb in the neonatal period.[20] With the decrease in pulmonary artery pressure and pulmonary vascular resistance that normally occurs in the first weeks of life,

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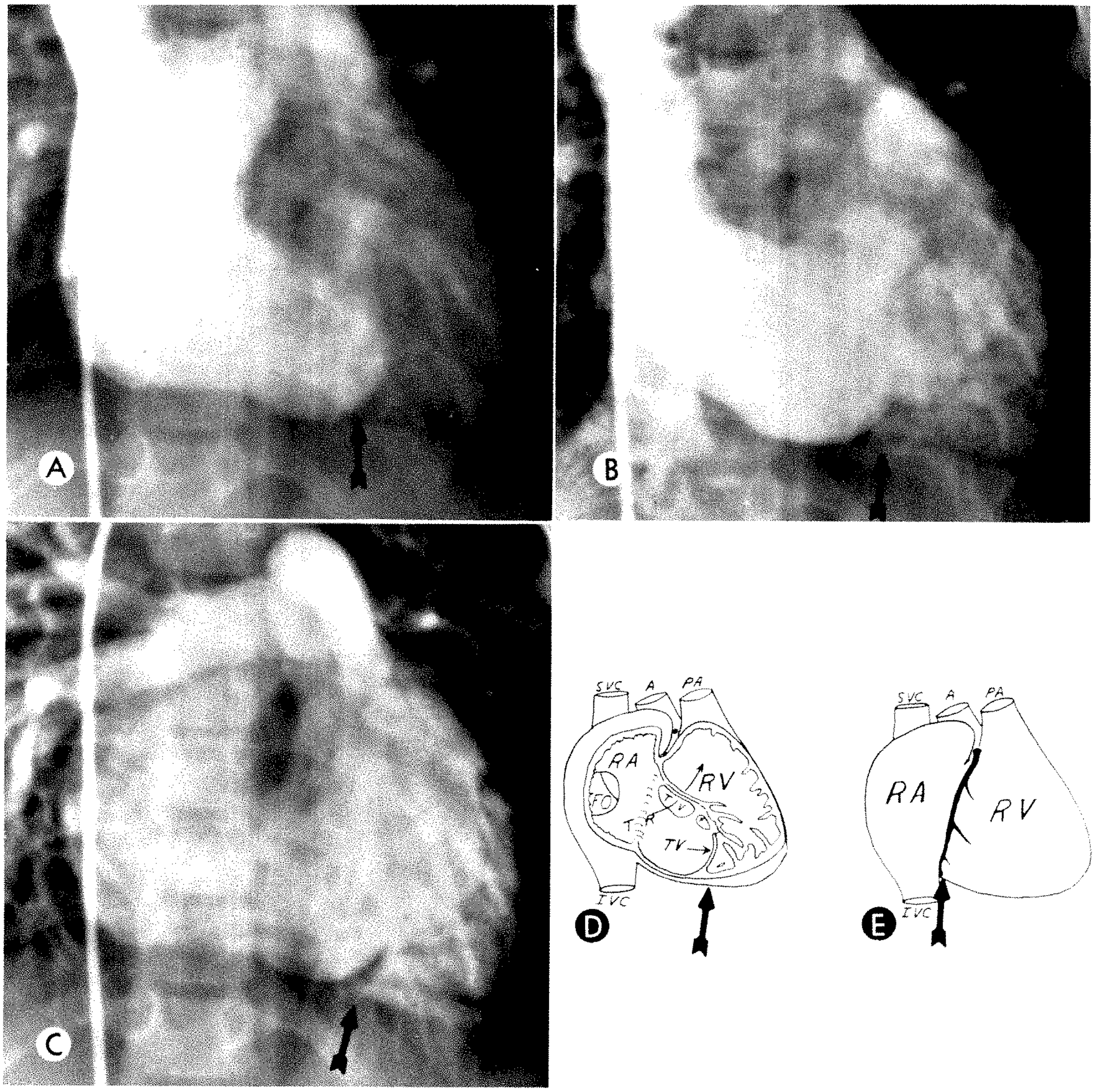

FIG. 1. Case 1. Twelve year old boy with cyanosis since infancy. (*A*) Frontal angiocardiogram with the right atrium opacified demonstrating extension of the right atrial cavity into the sinus portion of the right ventricle because of the downward displacement of the tricuspid valve leaflet (arrow). (*B*) Atrial systole demonstrating contraction of the right atrium with reflux of the radiopaque contrast material into the inferior vena cava, filling of the right ventricle and dilatation of the "atrialized portion" of the right ventricle. (*C*) One second later with both atrium and ventricle opacified, the displaced tricuspid valve is seen as a filling defect (arrow). (*D*) Diagram showing the displaced tricuspid valve (TV) apparatus (arrow) relative to the tricuspid valve ring (TR). (*E*) Diagram of the frontal surface of the heart showing the position of right coronary artery (arrow) in the right atrioventricular sulcus and its close relationship to the tricuspid valve ring as shown in *D*. RA = right atrium; RV = right ventricle; SVC = superior vena cava; A = aorta; PA = pulmonary artery; FO = foramen ovale; IVC = inferior vena cava. (The same symbols are used in Fig. 4*D*.)

the cyanosis tends to become less or to disappear and usually a long asymptomatic period begins.

As might be expected, incompetence of tricuspid valve closure is common either at the main orifice or by way of fenestrations which are frequently present in the deformed leaflets. When the tricuspid insufficiency is severe, marked dilatation of the functional right ventricular cavity sometimes occurs (Fig. 5, *A–E;* and 6, *A*, *B* and *C*).

CLINICAL FEATURES

Malfunction of the right heart is the basis for the clinical features of Ebstein's anomaly. Limited exercise tolerance and fatigue are common symptoms. The major clinical signs are cyanosis, arrhythmias and right heart failure. Longevity varies greatly depending upon the severity of the malformation, but the average survival is into young adult life. Death most often results from right heart failure or serious arrhythmias.

Heart murmurs are usually present, most commonly a systolic murmur which is heard best between the lower left sternal border and the apex. Diastolic sounds are also frequent and may produce a triple or quadruple rhythm or gallop that is believed by some observers to be characteristic.[3,7,10,14,18]

Electrocardiographic evidence of conduction defects (notably prolonged P-R interval and incomplete and complete right bundle branch block) and arrhythmias (especially paroxysmal supraventricular tachycardia and atrial fibrillation) are not surprising in view of the marked dilatation of the right atrium and the right ventricular deformity.

ROENTGENOGRAPHIC FEATURES

A clear understanding of the anatomy and pathologic physiology is essential for proper interpretation of the roentgenographic findings. Since the severity of the malformation varies greatly, the roentgenographic findings range from normal to severely abnormal.

Although in mild cases the heart may be normal in size, more typically cardiomegaly is present, often in severe degree. In the frontal projection, the enlarged heart may have a square[1] or block-like configuration (Fig. 2, *A* and *B*). The enlarged right atrium is responsible for the right heart border. A convex left upper heart border may be produced by a prominent right ventricular outflow tract pushed over by the right heart enlargement and by some clockwise rotation of the heart. This feature is made more conspicuous by the absence of main pulmonary artery enlargement, and can erroneously suggest enlargement of the left atrial appendage in the frontal projection.

In the lateral view, however, left atrial enlargement is not present—a valuable consideration in differential diagnosis. The enlarged right heart, especially the right atrium, increases the density of the retrosternal region.

The lung vessels, including the main pulmonary artery, are of either normal or decreased prominence, because the pulmonary artery pressures are normal, and right ventricular output is either normal or reduced. When marked cardiomegaly and small central and peripheral pulmonary vessels are visualized and are accompanied by clinical signs of right heart failure, the question of Ebstein's anomaly should be raised.

Not infrequently, the large right heart so displaces the left ventricle laterally and posteriorly that a configuration quite typical of left ventricular enlargement is produced on the frontal roentgenogram (Fig. 5, *A–E* and 6, *A*, *B* and *C*). The reduced prominence of the pulmonary vessels together with the electrocardiographic findings will usually warn of this potential error. This configuration most commonly occurs when the tricuspid insufficiency is severe and accompanied by enlargement of the functional right ventricle.

Cardiac pulsation is usually not prominent at fluoroscopy unless considerable tricuspid insufficiency is present, in which case vigorous pulsation may be observed.

ANGIOCARDIOGRAPHIC FEATURES

We have found angiocardiography valuable and often essential in making the diagnosis of Ebstein's anomaly. Selective injections of contrast material in the region of the tricuspid valve are usually employed. Occasionally, a left sided study is necessary to rule out abnormality of the left heart and to locate the right atrioventricular sulcus by demonstrating the position of the right coronary artery (Fig. 4, *A–F*). We have preferred serial roentgenography for the illustrations in this paper. Cine-angiocardiography can also clearly delineate the pathologic anatomy and can often better demonstrate the functional derangements in this anomaly.

Demonstration of the following features has been useful:

1. Downward displacement and deformity of the tricuspid valve leaflets.
2. Large right atrium with deformed, small, or occasionally large right ventricle.
3. Position of right coronary artery; by locating the right atrioventricular groove, and hence the approximate position of the tricuspid valve ring, helps to document abnormal displacement of the tricuspid leaflets.
4. Right to left interatrial shunt via patent foramen ovale or atrial septal defect.
5. Normal or small main pulmonary vessels.
6. Volume changes in the "atrialized" right ventricle corresponding to ventricular systole even though catheter pressures demonstrate this region to be in the functional right atrium.
7. Large reciprocal volume changes between functional right ventricle and right atrium when marked tricuspid insufficiency is present.
8. Normal sized or relatively small left heart structures and aorta.
9. Additional congenital cardiac malformations.

ILLUSTRATIVE CASES

Seven selected examples of Ebstein's anomaly follow. The ages of the patients at the time of study varied from 6 to 50 years. The first 4 cases demonstrate the range in severity of displacement of the tricuspid valve into the right ventricle. Cases v and vi demonstrate Ebstein's anomaly with severe tricuspid insufficiency. Case vii is a rare example of Ebstein's anomaly of the left atrioventricular valve.

Case i (Fig. 1, *A–E*). S.S. Unit #146-81-93. This 12 year old white boy was known to have a heart murmur and intermittent mild cyanosis since infancy. He was asymptomatic except for slightly reduced exercise tolerance.

Auscultation revealed a Grade 3/6 rough systolic murmur, maximal at the mid-left sternal border. A third heart sound was noted along the left sternal margin. The 2nd sound in the pulmonic area was mildly increased.

Electrocardiograms showed a prolonged P-R interval and complete right bundle branch block pattern.

Cardiac catheterization revealed a left-to-right shunt at the level of the superior vena cava. Peripheral arterial oxygen saturation was normal. There was slight elevation of the right atrial (mean 5 mm. Hg) and right ventricular (38/4 Hg) pressures with a small systolic pressure gradient across the pulmonary valve (pulmonary artery 21/7 Hg). A selective angiocardiogram from the pulmonary artery revealed partial anomalous venous return from both lungs to the superior vena cava. Selective angiocardiograms from the superior vena cava revealed Ebstein's anomaly of the tricuspid valve.

Death occurred unexpectedly 2 days after correction of the anomalous pulmonary venous drainage and closure of sinus venosus type interatrial communication.

The diagnosis of Ebstein's anomaly was confirmed at necropsy.

Comment. An exceptionally clear demonstration of Ebstein's anomaly of the tricuspid valve was provided by angiocardiography. In this case, there was an associated anomaly of pulmonary venous drainage.

Case ii (Fig. 2, *A* and *B*). S.M. Unit #148-17-31. This 19 year old Negro college boy was asymptomatic until onset of exercise-induced syncopal attacks at 16 years. There was no history of cyanosis.

Auscultation revealed a Grade 2/6 mid-systolic murmur, maximal at the low left sternal border and a Grade 2/6 mid-diastolic murmur at the apex. The 2nd sound in the pulmonic area was normally split and not accentuated. The

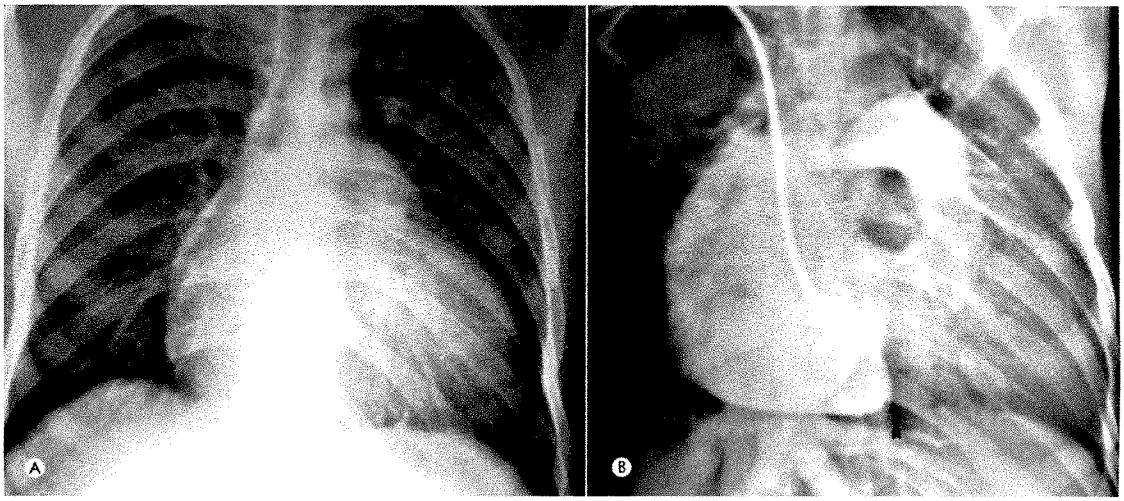

FIG. 2. Case II. Nineteen year old man with syncopal episodes. (*A*) Frontal chest roentgenogram demonstrating moderate cardiomegaly with "box-like" configuration due to prominent right atrial border and secondary prominence of the right ventricular out-flow tract along the left mid and upper heart border, mainly because of clockwise rotation of the heart secondary to the large right atrium. The pulmonary vasculature is normal. (*B*) Frontal angiocardiogram demonstrating the large opacified right atrium, deformed tricuspid valve leaflet (arrow) and relatively small residual right ventricular cavity. The tricuspid valve displacement is minimal in this case, but the diagnosis of Ebstein's anomaly is strongly supported by the intracardiac electrocardiogram.

first heart sound was widely split at the base and along the sternum.

Electrocardiograms showed a prolonged PR interval, left axis deviation, and marked intraventricular conduction defect (QRS interval =0.16 second).

Cardiac catheterization revealed severe elevation of right atrial mean pressure (15 mm. Hg) and right ventricular end-diastolic pressure (RV 26/15 mm. Hg and pulmonary artery 26/18 mm. Hg). The arterial oxygen saturation was normal. Simultaneous intracardiac electrocardiographic and pressure tracings on withdrawal through the tricuspid valve revealed an atrial pressure curve and a ventricular cavity electrocardiographic pattern consistent with downward displacement of the tricuspid valve into the right ventricular cavity. Selective angiocardiograms performed from the right atrium and pulmonary artery demonstrated an Ebstein's anomaly.

Comment. A typical case of Ebstein's anomaly with only mild displacement of the tricuspid valve into the right ventricle is illustrated. The marked right atrial enlargement and secondary clockwise rotation of the heart were mainly responsible for the block-like silhouette. The right ventricle is small and poorly compliant.

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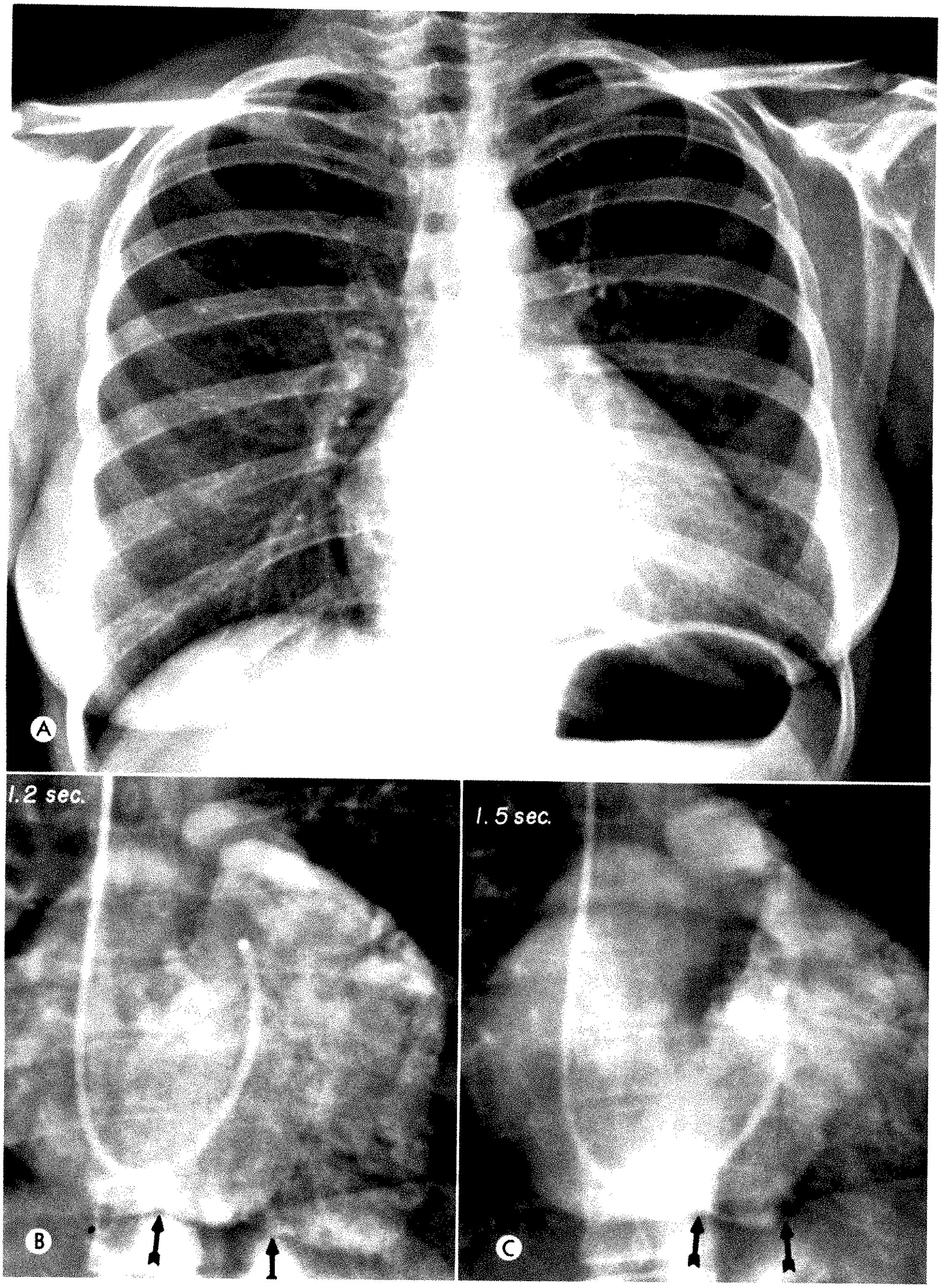
FIG. 3. Case III. Twenty year old cyanotic woman. (*A*) Frontal chest roentgenogram demonstrating moderate nonspecific cardiomegaly except for some prominence of the right atrial border. Pulmonary vascularity is normal. (*B*) Frontal angiocardiogram in ventricular diastole with the right heart opacified. The arrow on the patient's left points to the abnormally positioned tricuspid valve leaflet. The other arrow points to approximate position of the tricuspid valve ring. (*C*) Frontal angiocardiogram 0.3 second later in ventricular systole. The right ventricle is contracted and the pulmonary vessels are more distended. Note that the atrialized portion of the right ventricle (between the two arrows) has also contracted with ventricular systole, but that the rest of the right atrium is much larger because of this contraction of the "atrialized" right ventricle and perhaps also because of some insufficiency at the displaced tricuspid valve.


A
1.2 sec.
B
1.5 sec.
C

The arrhythmias associated with paroxysmal tachycardia, which produced syncope in this patient, were controlled by quinidine therapy.

Case iii (Fig. 3, *A*, *B* and *C*). K.H. Unit #97-78-31. This 20 year old white woman was cyanotic since birth, but this had lessened in intensity during childhood. Clubbing first appeared in adolescence. She has mild exercise intolerance.

Auscultation revealed a Grade 3/6 systolic murmur, maximal at the low left sternal border and a soft mid-diastolic murmur in the same area.

Electrocardiogram showed a right axis deviation and incomplete right bundle branch block pattern.

Cardiac catheterization indicated an elevated mean right atrial pressure (6 mm. Hg) and a small systolic pressure gradient (10 mm. Hg) between the right ventricle (25/5 mm. Hg) and pulmonary artery. Peripheral arterial oxygen saturation was 78 per cent. An intraventricular type intracardiac electrocardiographic pattern was recorded simultaneously with a right atrial pressure curve in the region just proximal to the effective tricuspid valve.

Comment. This is a typical case of Ebstein's anomaly associated with a right-to-left shunt at the atrial level and obvious peripheral arterial desaturation. The angiocardiogram clearly demonstrated the moderately displaced and deformed tricuspid valve. A small systolic pressure gradient across the pulmonic valve, as noted in this case, is not uncommon. The right ventricular end-diastolic pressure was very slightly diminished compared to that in the right atrium suggesting minimal obstruction to flow by the deformed tricuspid valve.

Case iv (Fig. 4, *A*-*F*). M.M. Unit #157-54-41. This 11 year old girl was noted to have a heart murmur and cyanosis in the neonatal period. Cyanosis diminished after several months and was noted only rarely since then. The child is asymptomatic except for easy fatigability.

Auscultation revealed a quadruple rhythm and Grade 3/6 harsh pansystolic murmur, maximal at the low left sternal border. The second sound in the pulmonic area was widely split.

Electrocardiograms showed an intraventricular conduction delay of the incomplete right bundle branch block type.

Cardiac catheterization revealed normal pressures and no evidence of intracardiac shunts. The catheter passed from the right to the left atrium.

Selective angiocardiograms were performed from the "atrialized portion" of the right ventricle and the left atrium, and showed an Ebstein's malformation of the tricuspid valve.

Comment. The diagnosis of Ebstein's anomaly in this patient was first suggested by angiocardiography. An example of severe displacement of the tricuspid valve into the right ventricle is illustrated with excellent angiographic demonstration of these findings. This is the first reported example of utilization of right coronary artery visualization to document the position of the right atrioventricular groove in the demonstration of abnormal displacement of the tricuspid valve into the right ventricle.

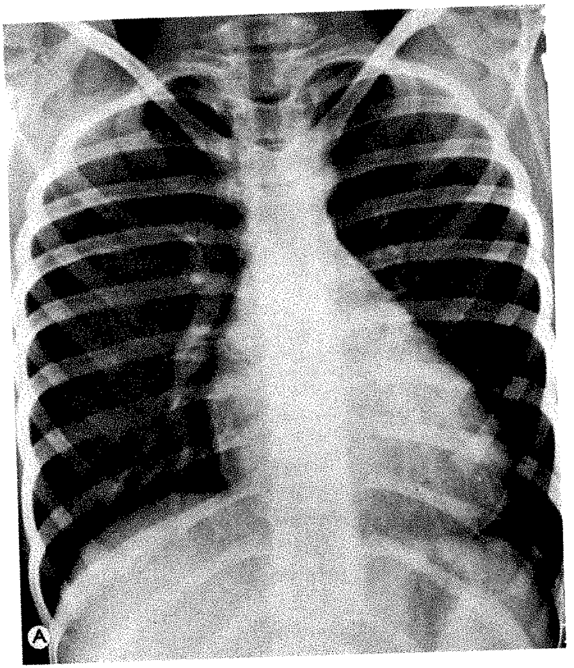


Fig. 4. Case iv. Eleven year old asymptomatic girl. (*A*) Frontal chest roentgenogram demonstrating a moderately large heart with a configuration which suggested left ventricular enlargement. The pulmonary vascularity is normal or less than normal in prominence and the main pulmonary artery is not enlarged.

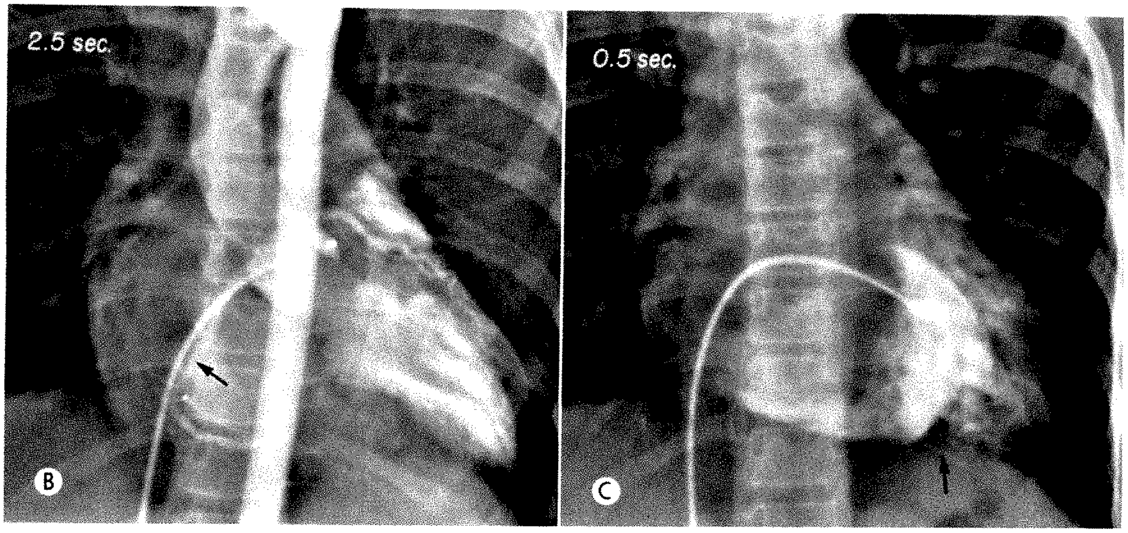


Fig. 4. (*B*) Frontal angiocardiogram 2.5 seconds following injection of radiopaque contrast material into left atrium demonstrates a relatively small left ventricle. Note particularly the position of the right coronary artery (arrow) which parallels the course of the catheter and establishes the approximate position of the tricuspid valve ring. (The catheter had traversed an atrial septal defect.) (*C*) Frontal angiocardiogram with injection of radiopaque contrast material through catheter, which by pressure record was in the right atrium. Note the unusual leftward extension of the right atrium. The arrow points to the displaced tricuspid valve leaflet. The large "atrialized" portion of the right ventricle extends from the approximate position of the right coronary artery (*B*) to the displaced valve leaflet.

Case v (Fig. 5, *A–E*). F.M. Unit #77-89-55. This 50 year old woman was noted to have a heart murmur at the age of 35 years. Increasing shortness of breath and cardiomegaly subsequently developed, complicated by a chronic sideropenic anemia with esophageal web formation. Frank right heart failure first became manifest at the age of 49. No cyanosis or clubbing has been observed.

Auscultation revealed a short soft Grade 2/6 systolic murmur at the low left sternal border and a louder apical mid-diastolic murmur in addition to an atrial fourth heart sound.

Electrocardiograms showed a sinus bradycardia and short runs of atrioventricular nodal escape. There was a right axis deviation and an incomplete right bundle branch block pattern.

Interpretation of the chest roentgenograms as showing left ventricular enlargement led to left heart catheterization. The left heart was normal, but angiocardiography demonstrated posterior displacement of the left heart by a huge right atrium and ventricle. Right heart catheterization revealed that the right atrial mean pressure was 10 mm. Hg and the right ventricular pressure was 28/9 mm. Hg. Angiocardiography demonstrated a large right heart, tricuspid insufficiency and a deformity of the tricuspid valve. In the region just proximal to the effective tricuspid valve, an intraventricular type of electrocardiographic tracing was

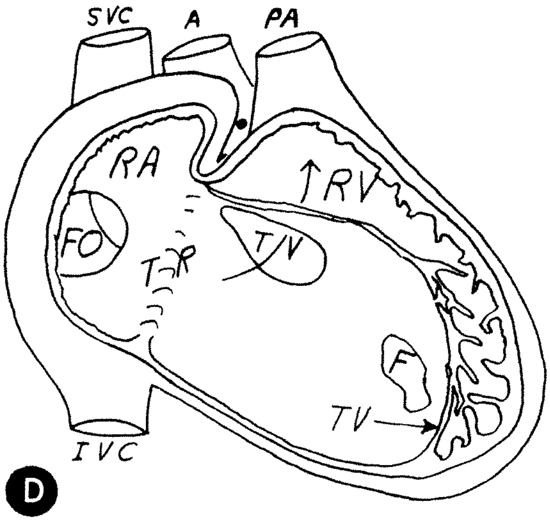


Fig. 4. (*D*) Diagram of right heart showing the marked deformity of the tricuspid valve as demonstrated in *C*. Note the marked downward displacement of the tricuspid valve (TV) relative to the tricuspid ring or annulus (TR). F = fenestration in deformed leaflet.

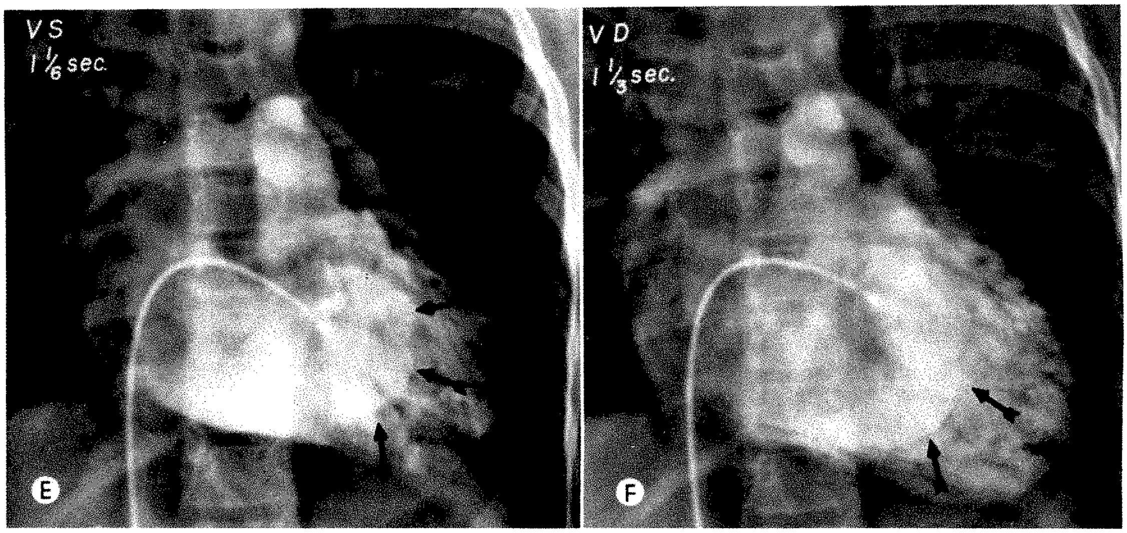


Fig. 4. (*E*) Frontal angiocardiogram 2/3 second after *C* showing that the right heart opacified in ventricular systole. The displaced tricuspid valve leaflets in the right ventricle are well shown (arrows). (*F*) Frontal angiocardiogram 1/6 second later in ventricular diastole. Nonopacified blood is diluting the radiopaque material in the right atrium.

obtained strongly supporting the diagnosis of Ebstein's anomaly.

Comment. This is an example of severe tricuspid insufficiency with normal pulmonary artery pressures. Simultaneous pressure tracings and intracardiac electrocardiography strongly support the diagnosis of Ebstein's anomaly. Severe tricuspid insufficiency with normal pulmonary artery pressures and in the absence of rheumatic heart disease is very rare, although when present Ebstein's anomaly may be most often responsible.[2,14,18]

Case vi (Fig. 6, *A*, *B* and *C*). J.W. Unit #133-92-31. This 8 year old boy was noted to be cyanotic after birth and developed signs of congestive heart failure on the first day of life. Cardiac catheterization at 1 month of age revealed a right ventricular pressure of 25/12 mm. Hg. Congenital pyloric stenosis was surgically cured at 2½ months. The boy was maintained on digitalis, and cyanosis has gradually receded. He has near normal exercise tolerance.

Auscultation reveals a Grade 3/6 harsh systolic murmur, maximal in the 4th left intercostal space and a Grade 2/6 mid-diastolic murmur in the same area.

Electrocardiograms show a prolonged PR interval, right axis deviation and a pattern of incomplete right bundle branch block.

Angiocardiography performed from the superior vena cava showed an Ebstein's anomaly of the tricuspid valve with marked tricuspid insufficiency.

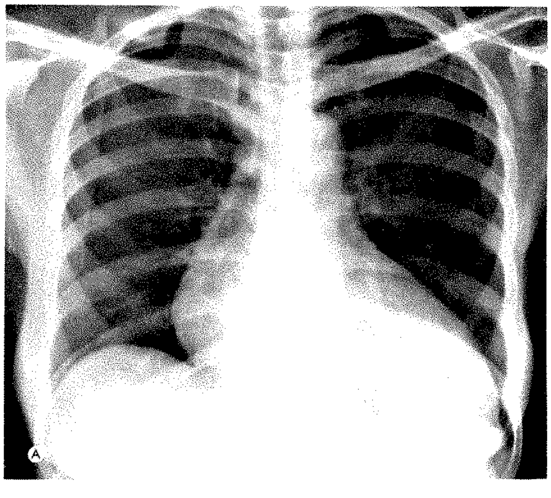


Fig. 5. Case v. Fifty year old woman in chronic right heart failure. (*A*) Frontal chest roentgenogram demonstrates marked cardiomegaly with a configuration suggesting left ventricular enlargement. Note the exceptionally translucent lung fields and lack of prominence of the central pulmonary arteries.

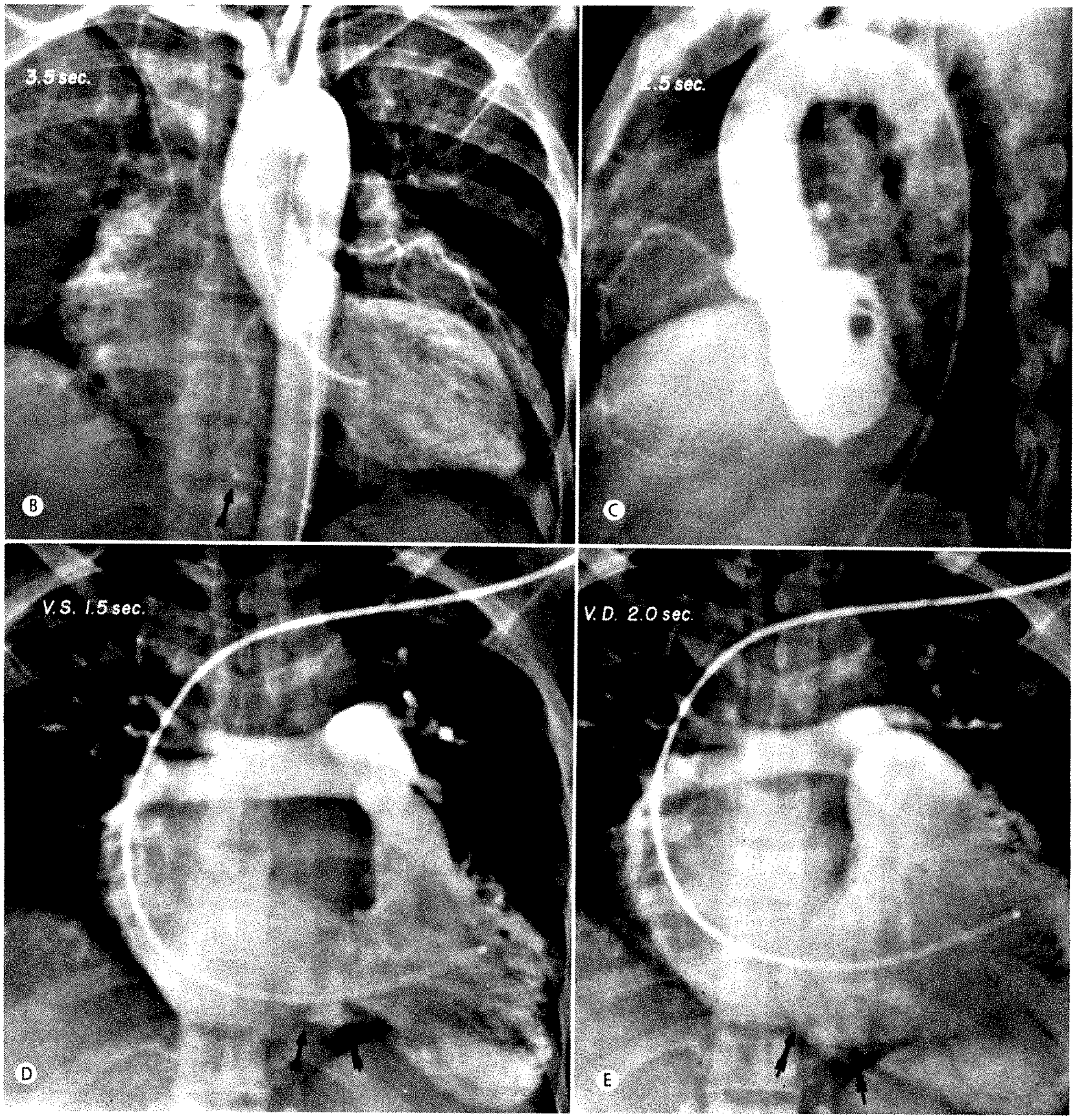
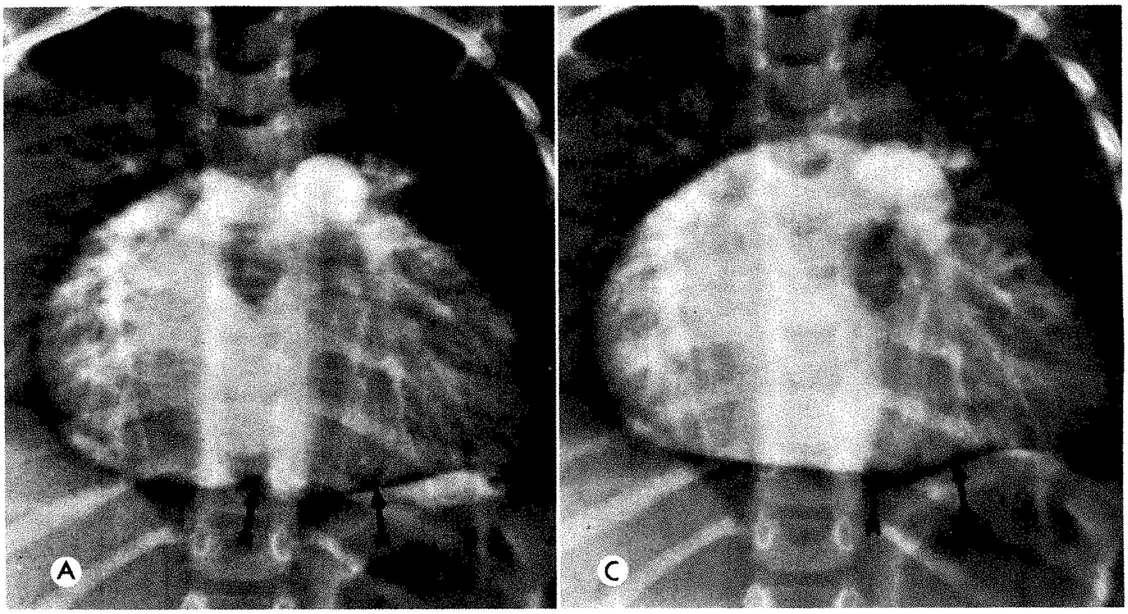


Fig. 5. (*B*) Frontal angiocardiogram following injection of radiopaque contrast material into the left ventricle demonstrates the size and position of the normal sized left ventricle. The arrow points to the position of the right coronary artery in the right atrioventricular groove (more medial arrow close to spine in *D* and *E*). (*C*) Lateral angiocardiogram showing the opacified left ventricle and aorta. The long distance between the left ventricle and right coronary artery suggests a large right ventricle. (*D*) Frontal angiocardiogram following injection of radiopaque contrast material into a large right ventricle showing, in ventricular systole, deformity of the tricuspid valve and considerable regurgitation of contrast material into right atrium. (*E*) Frontal angiocardiogram 0.5 second later showing the large size of the right ventricle in diastole. The right ventricle is so large that it extends to and forms the left border of the heart. Note the change in configuration of tricuspid valve region. The arrow on the patient's left points out the displaced tricuspid valve leaflet in this example of Ebstein's anomaly with severe tricuspid insufficiency.



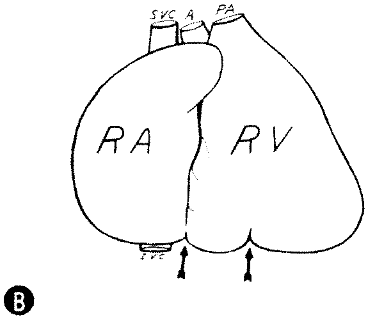


Fig. 6. Case vi. Eight year old boy with Ebstein's anomaly and severe tricuspid insufficiency. (*A*) Frontal angiocardiogram with right heart opacified in ventricular diastole demonstrates large right atrium and ventricle. (*B*) Diagram of *A*. In *A*, *B* and *C*, the arrow on the patients' left designates a displaced tricuspid valve leaflet and the other arrow points out the region of the tricuspid ring or annulus. (*C*) Frontal angiocardiogram in ventricular systole demonstrates contraction of the ventricle and simultaneously marked atrial dilatation, in part because of tricuspid insufficiency. Note that the "atrialized" portion of the right ventricle (between the arrows) contracts synchronously with the ventricle.

Comment. This case is an example of Ebstein's anomaly with impressive tricuspid insufficiency and considerable functional right ventricular enlargement in addition to marked right atrial dilatation. The "atrialized" portion of the right ventricle, contracting out of phase with the right atrium proper, contributes to the functional difficulty of the right atrium by acting like an atrial myocardial aneurysm or diverticulum.

Case vii (Fig. 7, *A–D*). T.R. Unit #136-14-40. This 6 year old boy was referred for evaluation at 1 year of age, at which time a heart murmur was first noted, because of intermittent wheezing episodes. Cyanosis was never observed except transiently on the first day of life. He is asymptomatic except for fatigue with exercise.

Auscultation revealed a Grade 4/6 pansystolic apical murmur and a Grade 3/6 mid-diastolic murmur at the apex.

Electrocardiograms showed intermittently a Wolff-Parkinson-White pattern. During normal sinus rhythm there was an intraventricular conduction defect of the incomplete left bundle branch block type.

Cardiac catheterization revealed an elevated pulmonary artery pressure (46/14 mm. Hg) but no intracardiac shunts.

A selective angiocardiogram from the right ventricle revealed congenitally corrected transposition of the great vessels with an anomaly of

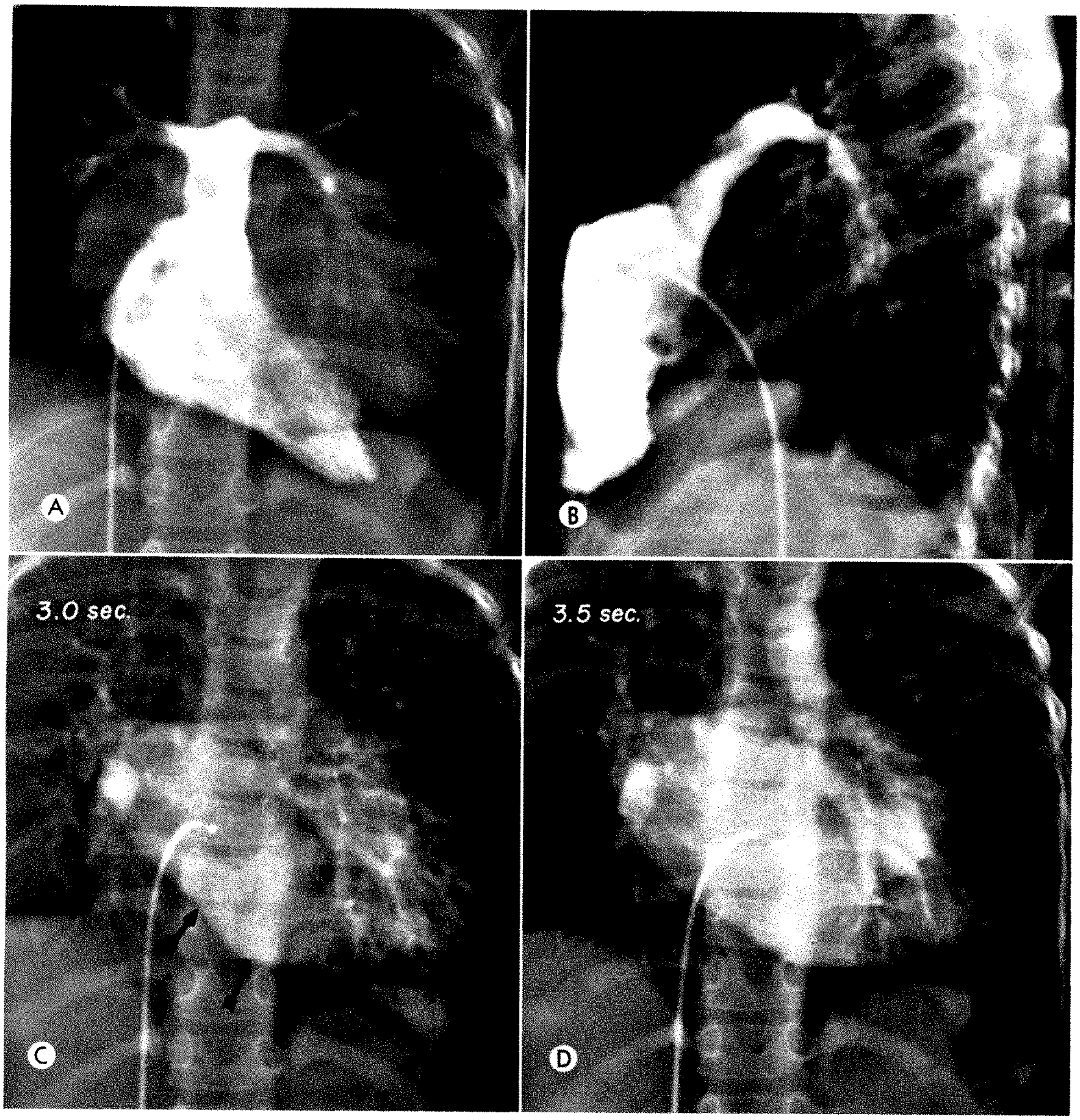


Fig. 7. Case VII. Five year old boy with Ebstein's anomaly of the left atrioventricular valve and congenitally corrected transposition of the great vessels. (*A*) Frontal angiocardiogram immediately following injection of radiopaque contrast material into the right-sided ventricle. Note the smooth wall of this ventricle (which has the anatomic structure of a left ventricle) and central position of the pulmonary artery origin. (*B*) Simultaneous lateral angiocardiogram demonstrating the relatively dorsal origin of the pulmonary artery from this ventricle. (*C*) Frontal angiocardiogram 2.5 seconds later showing opacification of the pulmonary veins, the left atrium and faintly the heavily trabeculated left-sided ventricle in diastole. Note the extension (arrows) of the left atrial cavity into this left-sided ventricle because of Ebstein's anomaly. (*D*) Frontal angiocardiogram 0.5 second later in ventricular systole showing the transposed position of the ascending aorta along the left upper heart border. Note the considerable increase in the size of the left atrium, in part because of insufficiency of the deformed left atrioventricular valve.

the left atrioventricular valve resulting in marked insufficiency and consistent with Ebstein's anomaly.

Comment. In congenitally corrected transposition of the great vessels, the left atrioventricular valve is anatomically like a tricuspid valve and the left sided ventricle is anatomically like a right ventricle. This, we believe, is the only situation in which Ebstein's anomaly is found at the left atrioventricular valve.[9,13,19]

Conduction defects are commonly noted on the electrocardiograms in patients with corrected transposition of the great vessels, irrespective of the presence or absence of associated anomalies. It is of interest that a Wolff-Parkinson-White pattern has frequently been documented in patients with Ebstein's malformation of the right sided tricuspid valve, but has not heretofore been reported when the left atrioventricular valve was involved.

DISCUSSION

Characteristic clinical and roentgenographic features will permit a diagnosis of Ebstein's anomaly with reasonable accuracy in many cases.[3,7,10,12,14] Confirmation of the diagnosis by cardiac catheterization and angiocardiography is important to rule out other lesions which may be currently amenable to corrective surgical procedures. Although no proven curative operative approach to Ebstein's anomaly is available, certain palliative surgical procedures, including anastomosis of the superior vena cava to the right pulmonary artery in order to bypass the right heart, have been successful in some cases.[20]

During cardiac catheterization, the large size of the right atrium is indicated by the wide loop made by the cardiac catheter in this chamber. The abnormal leftward extension of the right atrium and abnormal left sided position of the effective tricuspid valve (often to the left of the spine) can usually be appreciated. Right atrial and right ventricular end-diastolic pressures are typically elevated in contrast to the usually normal systolic pressure in the right ventricle and normal pulmonary artery pressures.

In 1954, Sodi-Pallares *et al.*[15] first suggested the usefulness of an electrode-tipped catheter for accurate identification by intracardiac electrocardiographic pattern of the chamber from which simultaneous pressures are being recorded. In Ebstein's anomaly, upon withdrawal of the catheter across the displaced tricuspid valve, an intraventricular electrocardiogram will be recorded simultaneously with an atrial pressure tracing from the "atrialized" portion of the right ventricle.[8] This technique proved definitely useful in 3 of the cases reported here. Transient arrhythmias, however, often occur as the catheter crosses the region of the tricuspid valve, making interpretation of both the electrocardiogram and the pressure contour difficult. Errors may occur when valve displacement is minimal or, theoretically, when stenotic tricuspid leaflets balloon into the right ventricle in the absence of Ebstein's anomaly.

Several deaths have been reported to be caused by cardiac catheterization or angiocardiography. Obviously, the risk of such procedures is likely to be higher in a condition frequently associated with arrhythmias. Nevertheless, in recent articles of large series of patients with Ebstein's anomaly, serious complications have not been reported.[3,10,14] Awareness of these risks, the immediate availability of efficient cardiac defibrillators, and the use of appropriate premedication, usually including quinidine or procaine amide (pronestyl), have undoubtedly made catheterization safer. Therefore, we believe that cardiac catheterization and angiocardiography in symptomatic individuals are warranted in the effort to establish a specific diagnosis of Ebstein's anomaly.

DIFFERENTIAL DIAGNOSIS

Ebstein's anomaly may be mimicked by other types of congenital heart disease, various myocardiopathies, and rheumatic heart disease. In patients with cyanosis and reduced prominence of the pulmonary vasculature, it is not surprising that in previous years there was occasionally diagnostic confusion with tetralogy of Fallot.[18] A more serious problem in differential diagnosis today, particularly in young children, is presented by severe pulmonary stenosis or atresia with intact ventricular septum and right to left interatrial shunt. The reduced pulmonary vascularity together with cardiomegally due to severe right heart dilatation when seen on chest roentgenograms may strongly resemble Ebstein's anomaly. Certain cases of tricuspid atresia may also provide a diagnostic problem in children.

Congenital tricuspid insufficiency not due to Ebstein's anomaly is very rare,[2] and unless a typical displacement deformity of the tricuspid leaflets can be demonstrated, the two possibilities cannot be distinguished short of direct inspection of the valve.

Ebstein's anomaly as a functionally important lesion is not regularly associated with other cardiac anomalies. Recently, however, Edwards[5] has demonstrated this lesion, at least in mild form, in most infants dying of pulmonary atresia with intact ventricular septum. As demonstrated in Case vii, Ebstein's anomaly of the left atrioventricular valve sometimes occurs in the presence of congenitally corrected transposition of the great vessels.[9,13,19]

When murmurs are inconspicuous, myocarditis, idiopathic myocardial hypertrophy, endocardial fibroelastosis and pericardial effusion become serious considerations in differential diagnosis. These conditions may usually be distinguished from Ebstein's anomaly by clinical features or angiocardiography. Pericarditis has been noted several times in Ebstein's anomaly as a terminal event, with a bloody pericardial effusion in at least 1 case.

In older patients without cyanosis, rheumatic heart disease with multi-valvular involvement or rarely isolated tricuspid valve disease may be serious diagnostic considerations.[3] In most of these cases left atrial enlargement would exclude the possibility of Ebstein's anomaly. Clinical tricuspid valve dysfunction may also result from carcinoid heart disease. When the cardiac configuration suggests left ventricular enlargement, a left parasternal systolic murmur is heard and the electrocardiogram is confusing, aortic stenosis may be incorrectly diagnosed.

SUMMARY

Angiocardiographic studies of 6 patients with Ebstein's anomaly of the tricuspid valve are presented to show variations in the extent of displacement of the tricuspid valve into the cavity of the right ventricle and the varying degrees of tricuspid insufficiency which may occur. In an additional patient, Ebstein's anomaly of the left atrioventricular valve was shown by angiocardiography.

Demonstration of the displaced tricuspid valve leaflets frequently makes possible a specific diagnosis of Ebstein's anomaly. Visualization of the right coronary artery in the right atrioventricular groove can help to locate the position of the tricuspid valve ring or annulus relative to the displaced leaflets.

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ARTERIOSCLEROTIC ANEURYSMS OF THE THORACIC AORTA*

INTRAVENOUS AND SELECTIVE VISUALIZATION

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VISUALIZATION of the thoracic aneurysms became practical with publication of the method of angiocardiography in 1938 and 1939.[8,9] Angiocardiography, by differentiating mediastinal tumors from vascular lesions, has made exploratory thoracotomy for the diagnosis of aneurysm obsolete.[10,11,23,25] Present-day bypass techniques, by permitting the resection of thoracic aneurysms and replacement with a prosthesis, have made the treatment of such lesions possible.[2,3,5,6] In this paper, illustrative examples of arteriosclerotic thoracic aneurysms visualized chiefly with the intravenous method of angiography are presented. Complicating lesions of the cardiovascular system, such as heart failure and pulmonary thrombosis, were readily demonstrated. Selective aortographic techniques for additional and special information concerning the brachiocephalic arteries, the abdominal aorta, and renal arteries were also obtained in some instances. These data comprise this report.

METHOD AND MATERIAL

Intravenous angiocardiography was performed according to the method previously described.[4,9] A large 12 gauge special needle stop-cock unit was inserted either percutaneously or by cut-down into a large vein of the arm. A preliminary circulation time determination with decholin (sodium dehydrocholate, 20 per cent) was then made.[18,19] Concentrated nonviscid contrast medium (usually angio-conray, iothalamic acid, 80 per cent),[21] 1 ml./per kg. of body weight was injected into a large vein of the arm with the Gidlund automatic pressure device at a pressure of 5 kg./cm.^{2}.[24] Serial roentgenography, with the patient supine, in simultaneous biplane (frontal and lateral) views was performed with the Schönander apparatus at the rate of one film per second for the duration of the circulation time. In very heavy individuals or when there was extreme cardiac enlargement owing to heart failure, angiocardiographic studies were first made in the frontal view and immediately followed by serial roentgenography in the left anterior oblique position. Selective aortograms were obtained following percutaneous transfemoral or transaxillary catheter injection of dilute contrast medium (20 to 40 ml.) with the Gidlund pressure apparatus at a pressure of 8 kg./cm.^{2}.[7] Rapid serial roentgenography (2 films per second) was performed, usually in the oblique or lateral positions, also with the Schönander apparatus.

Five cases of arteriosclerotic thoracic aneurysms, 1 woman and 4 men, ranging in ages from 53 years (the woman) to 76 years (Fig. 1 to 5), are presented. All but one were Caucasian and all had negative serologic tests for syphilis. Four patients had intravenous angiocardiography; in 2 this was supplemented by selective aortography. In 1 patient aortography to better define a descending thoracic aortic aneurysm could not be performed because of tortuosity of one of the iliofemoral arteries and occlusion of the other vessel. One of the 4 patients had a huge expansile abdominal aortic aneurysm which could not be visualized with the intravenous method because of heart failure and extreme dilatation of the entire thoracic aorta. Another patient had an isolated arteriosclerotic saccular aneurysm of the descending thoracic aorta which was visualized very satisfactorily with the technique of percutaneous retrograde transfemoral aortography.

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CASE STUDIES

Case 1. Conventional roentgenograms (Fig. 1, *A* and *B*) demonstrated a huge descending aortic arteriosclerotic aneurysm in a 61 year old man who complained of dyspnea and left chest pain. Because of increased venous pressure, obstruction of the great veins was suspected. However, intravenous angiocardiography showed an intact, dilated, and displaced right innominate vein and superior vena cava (Fig. 1*C*). Later in the series, there was reflux filling of the azygos vein. The right atrium, ventricle, and pulmonary artery were slightly enlarged. The right pulmonary artery was normal, but the left pulmonary artery was distorted (Fig. 1*D*). The left ventricular wall was found to be enlarged on a later film while the ascending aorta was dilated, measuring 50 mm. in mid-ascending portion (upper limit of normal, 38 mm.) (Fig. 1, *E* and *G*). A huge saccular aneurysm measuring 12 cm. in its opacified diameter and with a 6 cm. thrombosed outer wall was visualized later in the serial study (Fig. 1, *F* and *H*). The markedly tortuous brachiocephalic arteries and the origin of the saccular aneurysm well beyond the left subclavian artery were well demonstrated in the axillary selective aortogram (Fig. 1*I*). The aneurysmally dilated and tortuous terminal portion of the thoracoabdominal aorta was also strikingly shown in the retrograde abdominal aortogram (Fig. 1*J*).

Lack of pulsation of the aortic mass (Fig. 1, *A* and *B*) and its dense rounded character were consistent with the diagnosis of a posterior mediastinal tumor. Increase of venous pressure, believed to be due to obstruction of the great veins, was found, after intravenous angiocardi-

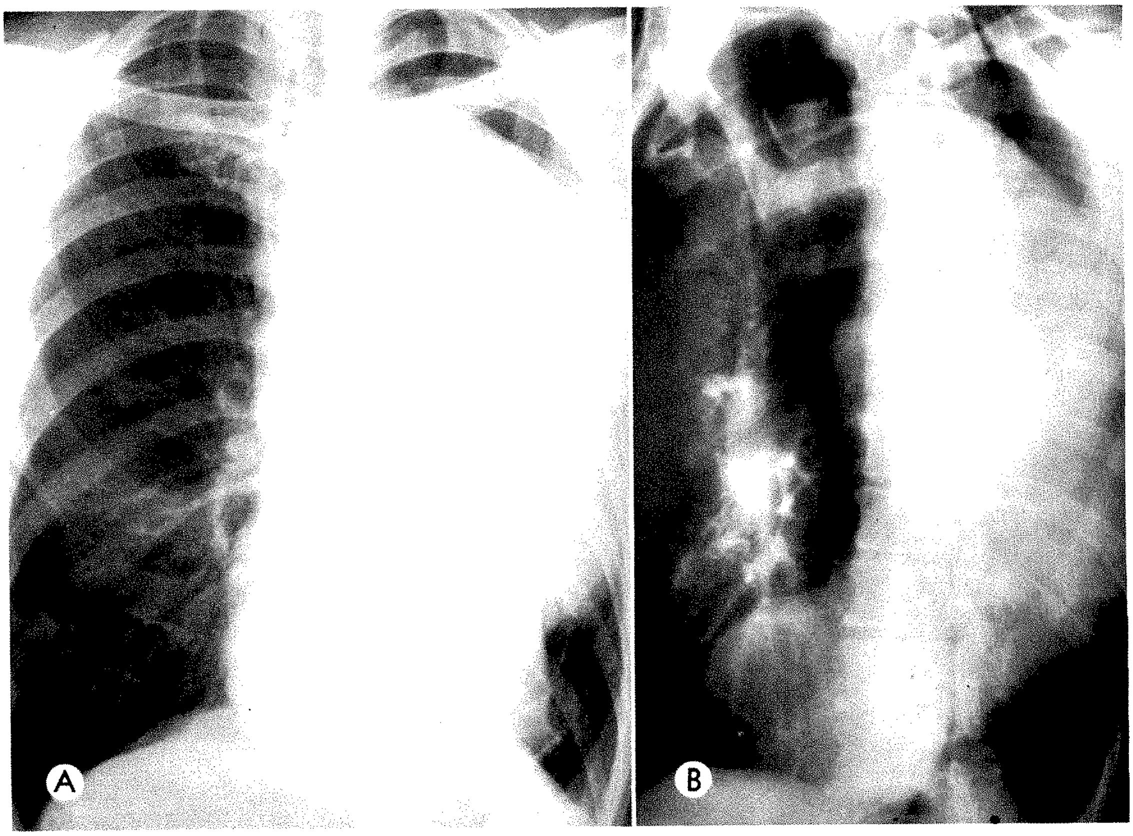


Fig. 1. Case 1. *Arteriosclerotic thoracic aneurysm in a 61 year old Negro with congestive heart failure.* (*A*) Frontal teleroentgenogram showing a huge rounded mass obscuring most of the left hemithorax. (*B*) Left oblique roentgenogram of the chest demonstrating the hugely dilated aortic arch and descending aorta; note the tortuosity and dilatation of the descending aorta at the diaphragm.

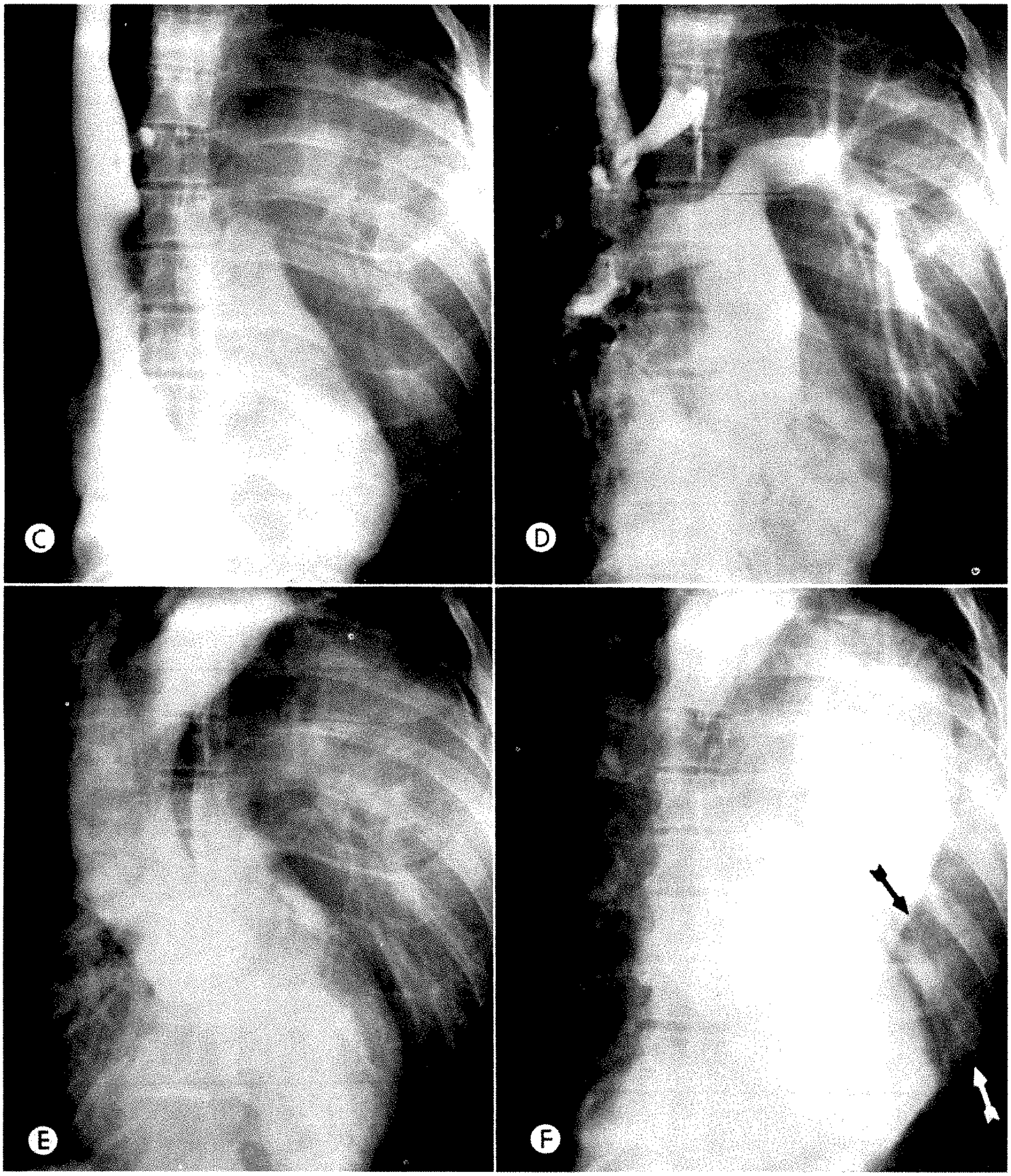


FIG. 1. (*C*) Frontal angiocardiogram at 2 seconds after beginning of injection showing the dilated and displaced superior vena cava. (*D*) Frontal angiocardiogram at 7 seconds after beginning of injection revealing the enlarged right ventricle and pulmonary artery. Note the distortion of the left pulmonary artery by the mass. (*E*) Frontal angiocardiogram at 13 seconds showing the dilated and tortuous but intact ascending aorta and brachiocephalic arteries. (*F*) Frontal angiocardiogram later in the series at 24 seconds showing the hugely opacified sacciform aneurysm with a thick thrombotic outer wall (bracketed by arrows).

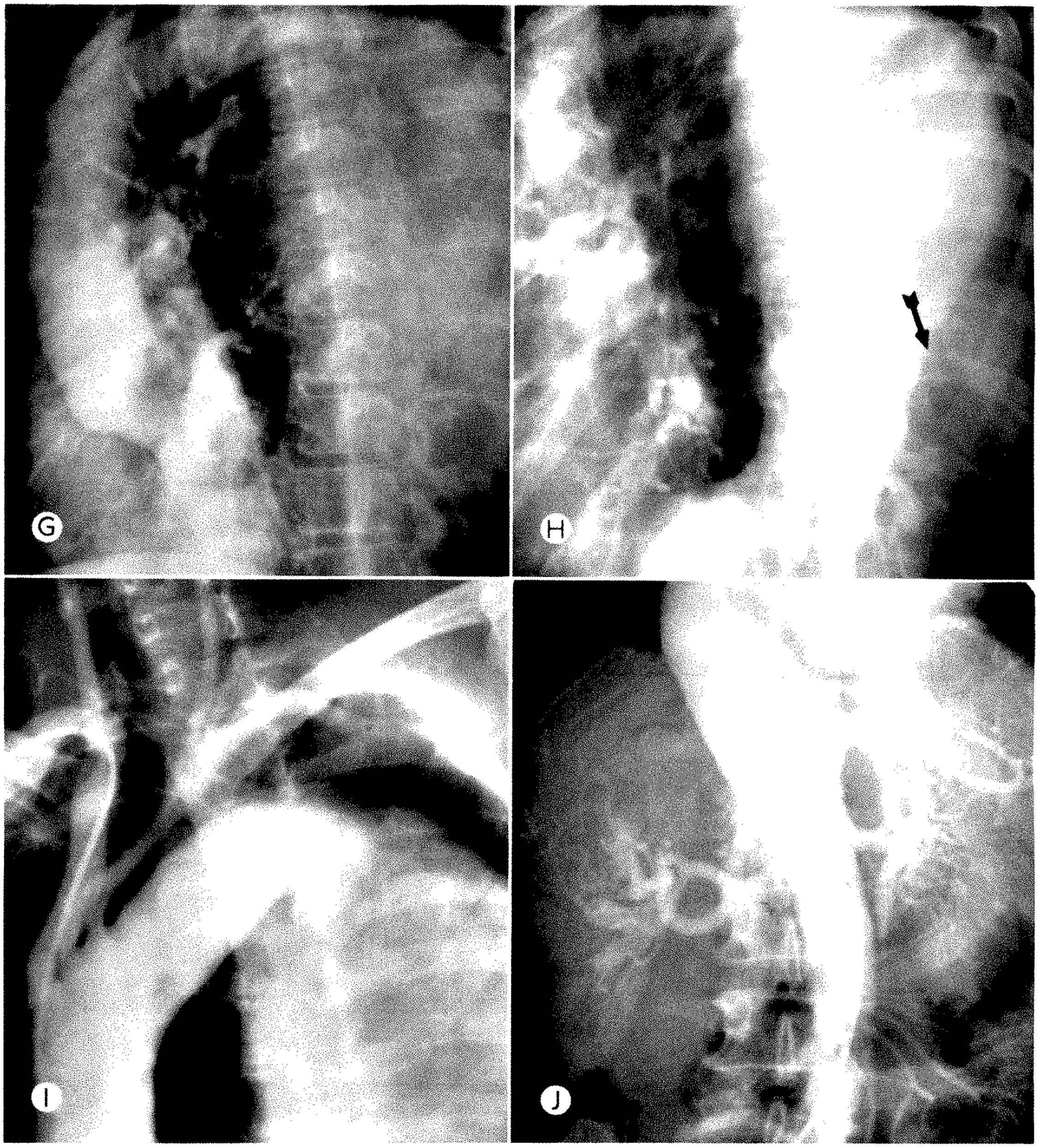


Fig. 1. (*G*) Left anterior oblique angiocardiogram at 13 seconds also showing the tortuous, unfolded, and intact ascending aorta and brachiocephalic arteries. (*H*) Left anterior oblique angiocardiogram at 24 seconds revealing the huge descending aortic aneurysm with its thick outer thrombotic wall (arrow). Note also the markedly dilated tortuous descending aorta at the diaphragm. (*I*) Left anterior oblique percutaneous right brachial retrograde aortogram showing the smooth, dilated, and tortuous character of the ascending aorta and brachiocephalic arteries. (*J*) Abdominal aortogram following percutaneous transfemoral injection of contrast medium demonstrating intact renal arteries with marked dilatation of the thoraco-abdominal aorta.

ography, to be due to chronic heart failure in this patient who had a prolonged circulation time (25 sec.), basilar rales, and a previous small left pleural effusion. Of interest also was the distortion of the left pulmonary artery by the aneurysm (Fig. 1*D*). The aortic aneurysm was huge and lined by thick thrombus (Fig. 1, *F* and *H*). Because of the marked dilatation and

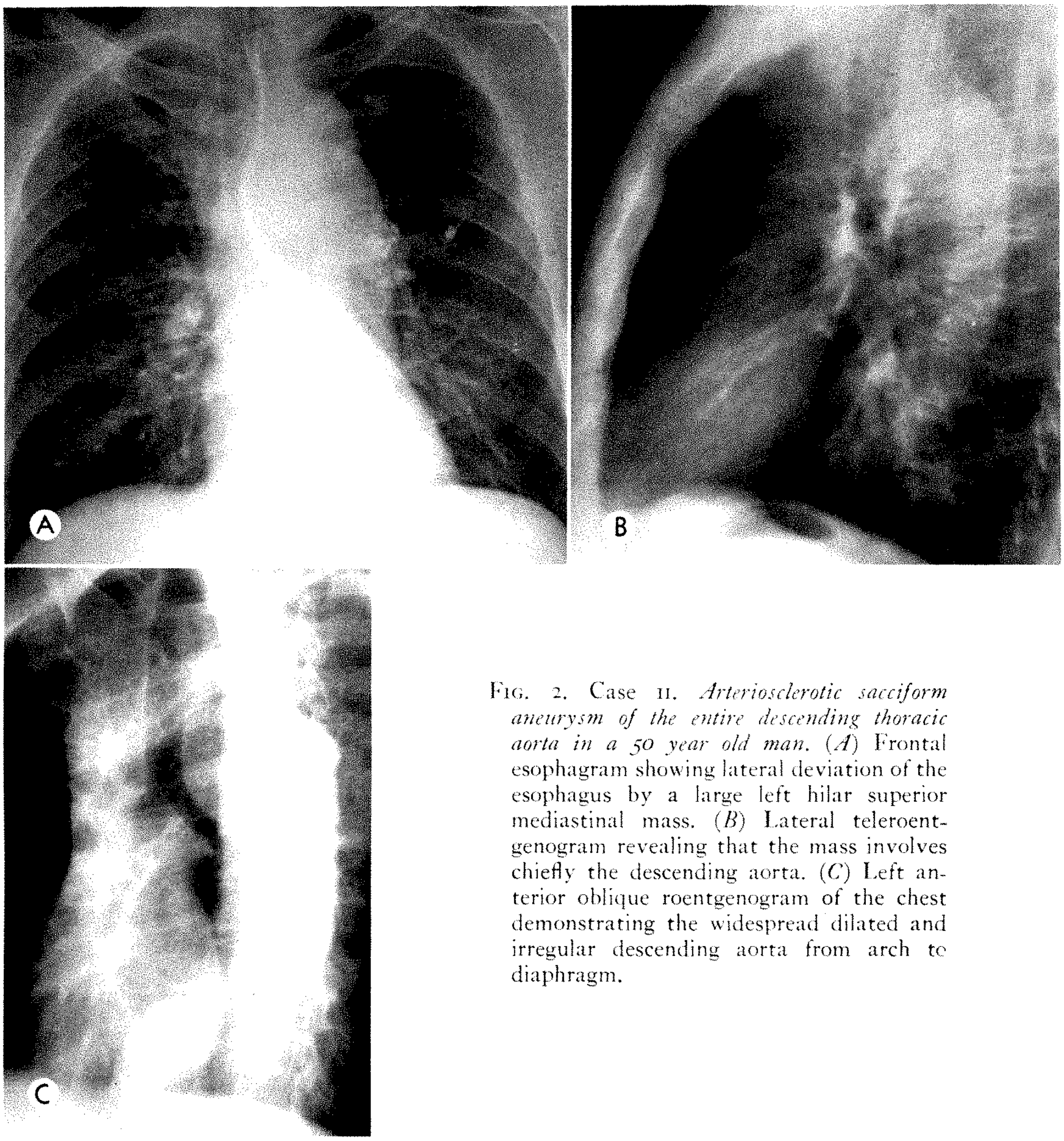
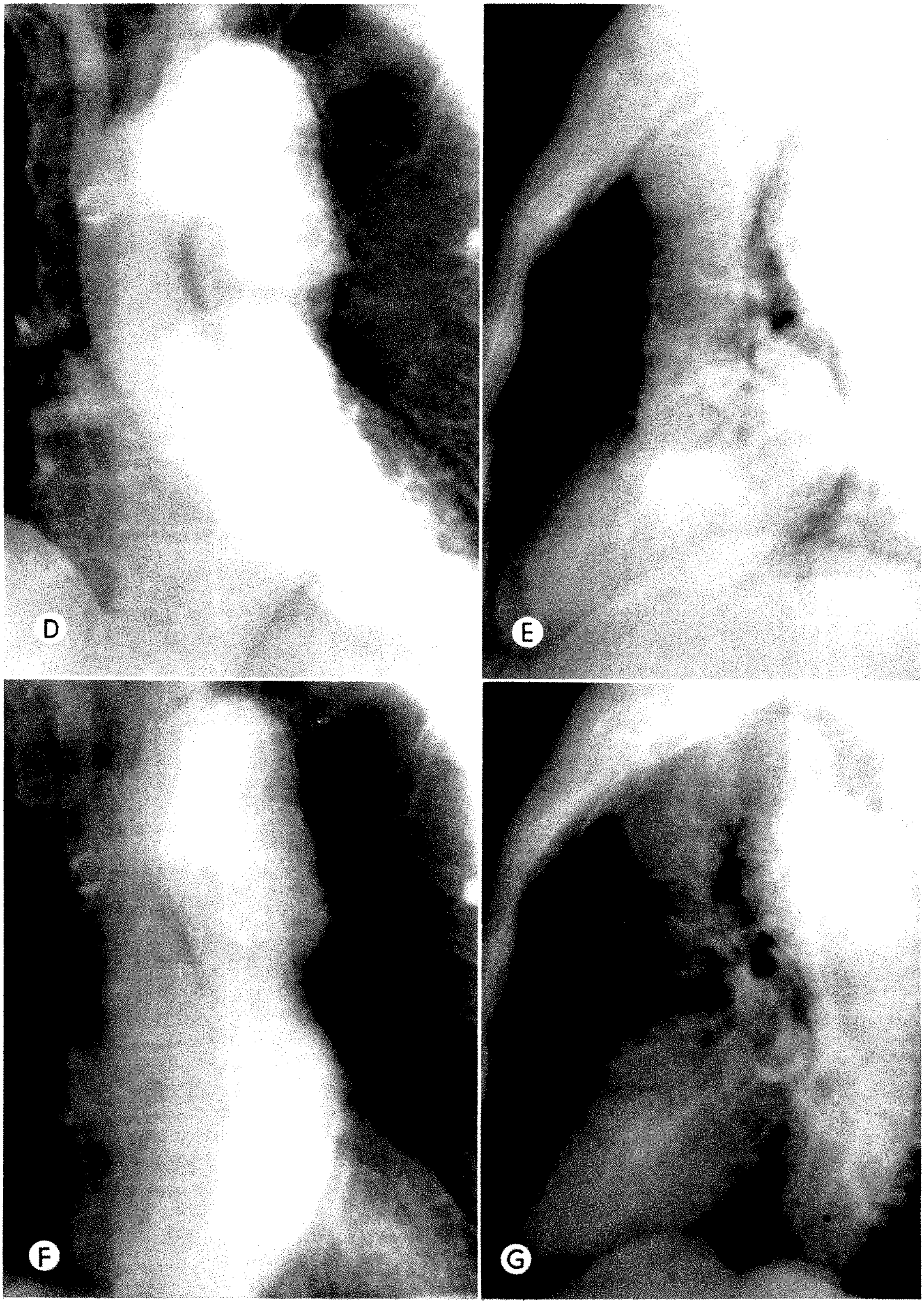


Fig. 2. Case II. *Arteriosclerotic sacciform aneurysm of the entire descending thoracic aorta in a 50 year old man.* (*A*) Frontal esophagram showing lateral deviation of the esophagus by a large left hilar superior mediastinal mass. (*B*) Lateral teleroentgenogram revealing that the mass involves chiefly the descending aorta. (*C*) Left anterior oblique roentgenogram of the chest demonstrating the widespread dilated and irregular descending aorta from arch to diaphragm.

tortuosity of the terminal end of the aneurysm (Fig. 1, *F* and *H*), a selective study of this portion of the vessel was performed in order to determine if there was also aneurysmal involvement of the abdominal aorta. Accordingly, percutaneous right axillary aortography was done and good visualization of the intact ascending aorta and brachiocephalic arteries was obtained (Fig. 1*I*); however, because of the increased size of the descending aortic aneurysm, insufficient contrast medium reached the abdominal aorta. Another approach via the percutaneous transfemoral route was made, and the widely dilated abdominal aorta proximal to the renal arteries was visualized (Fig. 1*J*).

Case II. A 50 year old man with a huge arteriosclerotic descending aortic aneurysm, which was discovered after left chest pain, was found to have a normal-sized heart, but with a markedly enlarged thoracic aorta (Fig. 2, *A*, *B* and *C*). Simultaneous biplane intravenous angiocardiography revealed an aneurysm of the entire descending aorta (Fig. 2, *D*, *E*, *F* and *G*). Retrograde transfemoral thoracic aortograms confirmed these findings (Fig. 2*H*) and also demon-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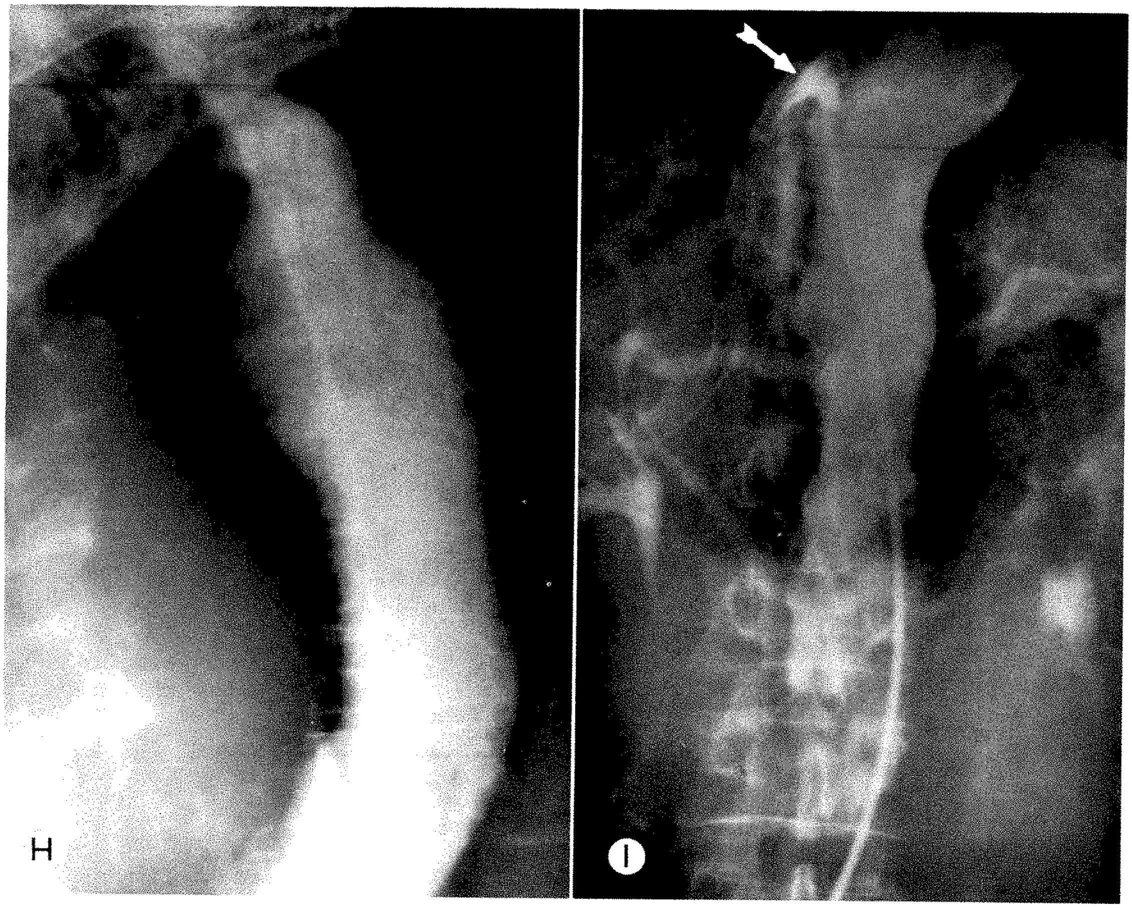

FIG. 2. (*H*) Thoracic aortogram obtained via a percutaneous transfemoral catheter shows the details of the descending aortic aneurysm to advantage. (*I*) Abdominal aortogram, made soon after, shows intact renal arteries, and aneurysmal and thrombotic disease of the abdominal aorta. Note the extravasation of a small quantity of contrast medium (arrow) into the wall of the abdominal aorta, an insignificant complication of the catheter injection technique.

strated arteriosclerotic thrombotic and aneurysmal disease of the entire abdominal aorta (Fig. 2*I*). Because of the normal-sized heart and circulation time (10 seconds) (Fig. 2, *A*, *B* and *C*), the cardiovascular system of this patient was studied, using simultaneous biplane intravenous angiocardiography.[20,24] The large aneurysm of the entire descending thoracic aorta was readily visualized (Fig. 2, *D*, *E*, *F* and *G*). The next day percutaneous retrograde femoral aortography was performed in order to examine in detail the markedly dilated descending aorta at the diaphragmatic level. This verified the intravenous angiocardiographic findings (Fig. 2*H*). Aneurysmal arteriosclerotic disease of the abdominal aorta was also demonstrated

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FIG. 2. (*D*) Frontal angiocardiogram showing the dilated but smooth ascending aorta and the large saccular aneurysm of the descending aorta. (*E*) Lateral angiocardiogram, simultaneous biplane of *D*, also revealing the dilated ascending aorta and aneurysmally dilated descending aorta. (*F*) Frontal angiocardiogram later in the series showing the large sacciform aneurysm of the upper portion of the descending aorta with the tortuous and dilated descending aorta. (*G*) Lateral angiocardiogram, simultaneous biplane of *F*, also showing the descending aortic aneurysm.

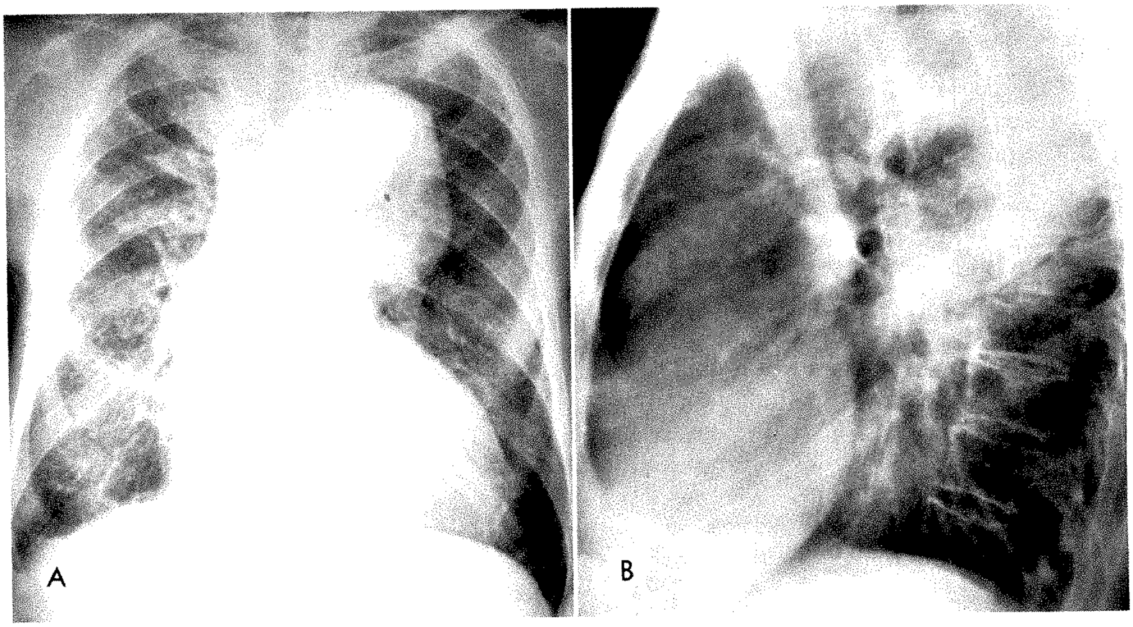

FIG. 3. Case III. *Aneurysmal dilatation of the entire thoracic aorta in a 76 year old man with aortic regurgitation, heart failure, hypertension (190/90 mm. Hg), intermittent claudication of both legs, and a huge abdominal aortic aneurysm.* (*A*) Frontal teleroentgenogram showing cardiac and aortic enlargement. (*B*) Lateral teleroentgenogram of the chest disclosing the large, tortuous, and dilated thoracic aorta.

(Fig. 2*F*). An unusual complication, subintimal extravasation of the contrast material into the aortic wall, apparently occurred during the selective procedure (Fig. 2*G*), but no untoward effect was noted.

CASE III. A 76 year old man with an enlarged heart and aortic arch (Fig. 3, *A* and *B*) complained of dyspnea, edema of the legs, and intermittent claudication of the legs. A huge pulsatile mass was palpable in the abdomen and

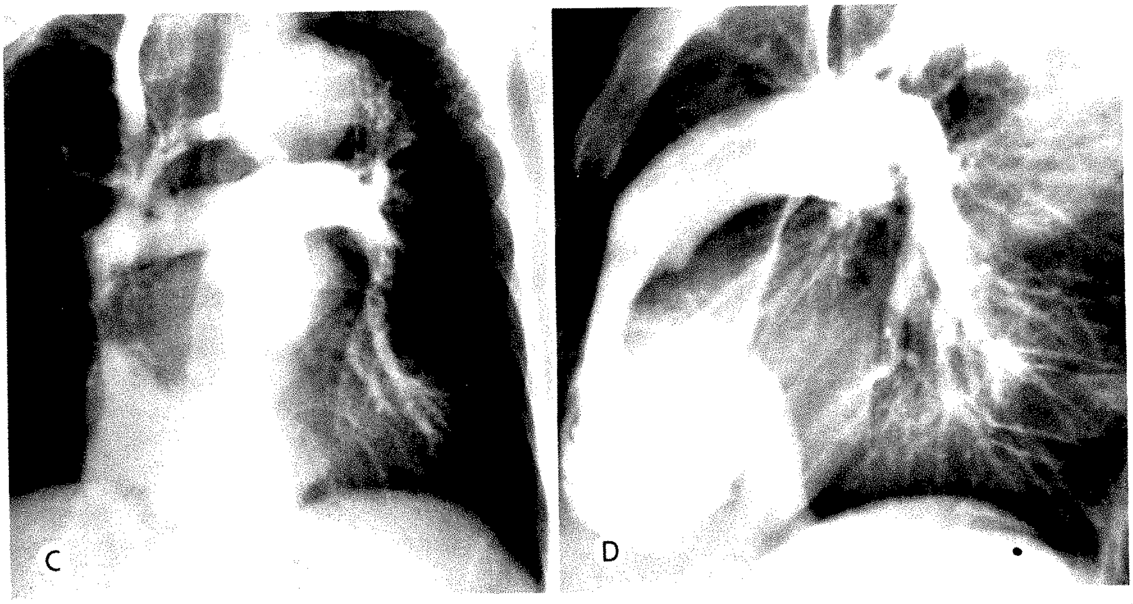

FIG. 3. (*C*) Frontal angiocardiogram at 4 seconds revealing retrograde filling of the azygos vein, enlargement of the right ventricle and pulmonary artery. (*D*) Lateral angiocardiogram, biplane study of *C*, also disclosing the right heart enlargement.

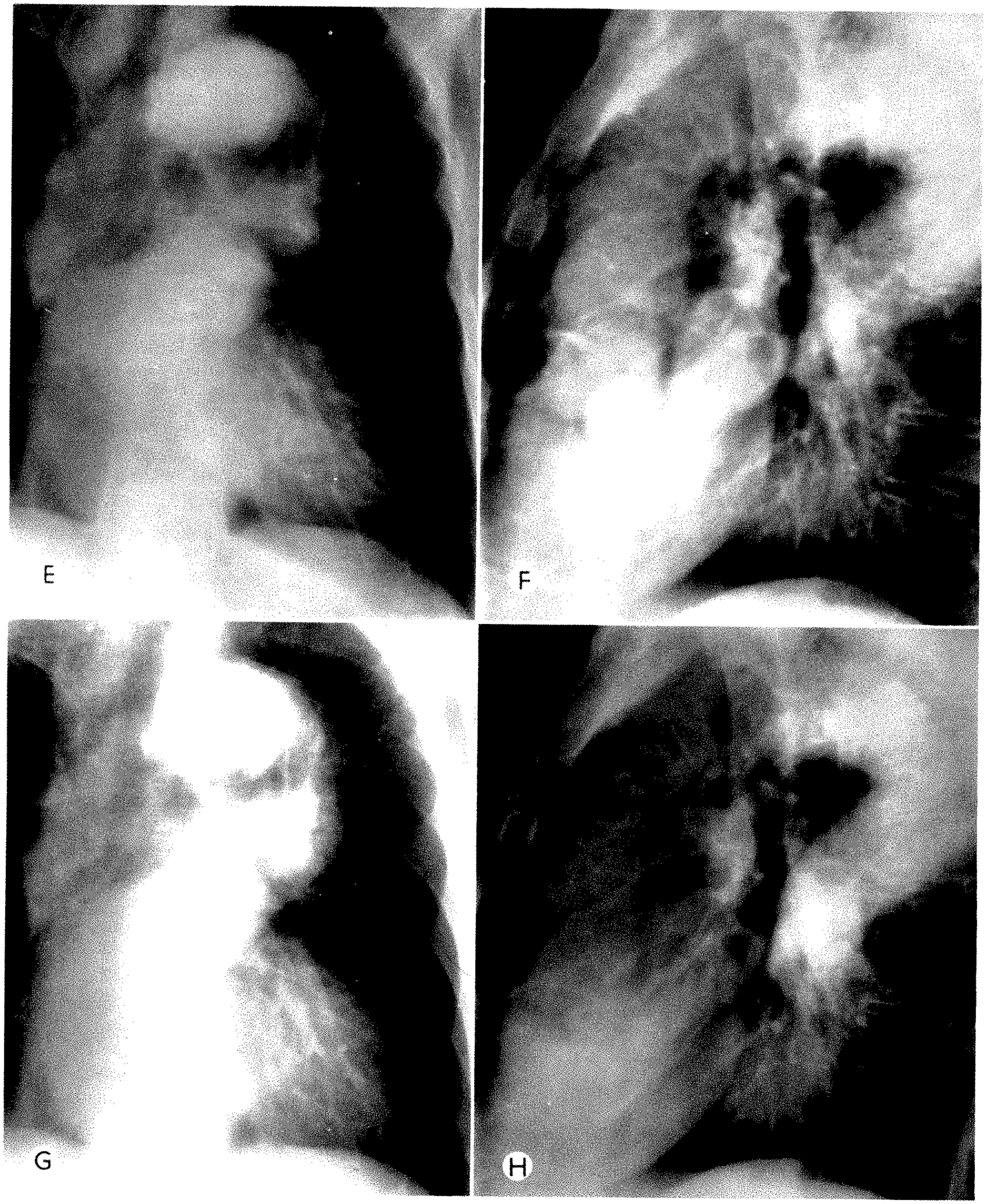

Fig. 3. (*E*) Frontal angiocardiogram at 10 seconds revealing the dilated ascending aorta and the dilated, tortuous, and unfolded aortic arch and descending aorta. (*F*) Lateral angiocardiogram, biplane study of *E*, also showing the aneurysmally dilated, tortuous, and unfolded ascending aorta, aortic arch, and descending aorta. (*G*) Frontal angiocardiogram at 24 seconds showing the aneurysmally dilated and tortuous descending aorta. (*H*) Lateral angiocardiogram, biplane study of *G*, showing that the aneurysmally dilated descending aorta reaches the level of the diaphragm.

there was aortic regurgitation and hypertension (190/90 mm. Hg). Biplane angiocardiograms demonstrated retrograde filling of the azygos vein with displacement of the right innominate vein and superior vena cava. The right atrium, ventricle, pulmonary artery, and branches were slightly enlarged (Fig. 3, *C* and *D*). The entire thoracic aorta was hugely dilated, measuring 65 mm. in the mid-ascending portion (average upper limit of normal, 38 mm.), 45 mm. beyond the left subclavian artery and 40 mm. at the level of the diaphragm (Fig. 3, *E*, *F*, *G* and *H*).

Initially, intravenous abdominal aortography was performed in order to visualize the pulsatile abdominal aortic aneurysm. The circulation time (decholin) was 20 seconds and even though abdominal films were exposed as late as 28 seconds after the beginning of injection, the abdominal aorta was not opacified. Therefore, simultaneous biplane angiocardiography at 1 second intervals for 25 seconds was performed and the enlargement of the right heart chambers was demonstrated (Fig. 3, *C* and *D*). The entire thoracic aorta was markedly tortuous and dilated (Fig. 3, *E*, *F*, *G* and *H*). Percutaneous axillary aortography was recommended for the visualization of the abdominal aortic aneurysm since both femoral and peripheral pulses were markedly diminished and because of the patient's complaints of intermittent claudication. This was not done because the patient was considered to be inoperable by his physician.

Case iv. A 63 year old man was admitted with congestive heart failure, infarction of the right lower lung, a huge heart, and a left hilar mass (Fig. 4, *A* and *B*). At first, he was suspected of having an aortic dissection; however, the localized character of the left hilus lesion (Fig. 4, *A* and *B*) made this diagnosis unlikely. Subsequently, hemoptysis, severe right chest pain, and right lower lung field infiltration were found to be caused by pulmonary emboli. Anticoagulant therapy was instituted and resulted in marked improvement of the patient. Following this, intravenous angiocardiograms, in frontal and left anterior oblique views, demonstrated thrombosis of the descending branches of the right pulmonary artery (Fig. 4, *C* and *E*), generalized cardiac enlargement and a localized aneurysm of the descending aorta (Fig. 4, *D* and *F*). Because of the cardiac dilatation and enlargement due to congestive heart failure (circulation time [decholin] 26 seconds), there was poor visualization of the descending aorta. Percutaneous retrograde femoral aortography for confirmation could not be performed because of severe tortuosity of the right iliofemoral artery and occlusion of the left iliofemoral artery. Permission for transaxillary selective aortography was not granted by the patient.

Case v. This 53 year old woman experienced left scapular pain for a period of 3 months. Because the femoral pulses were vigorous, it was

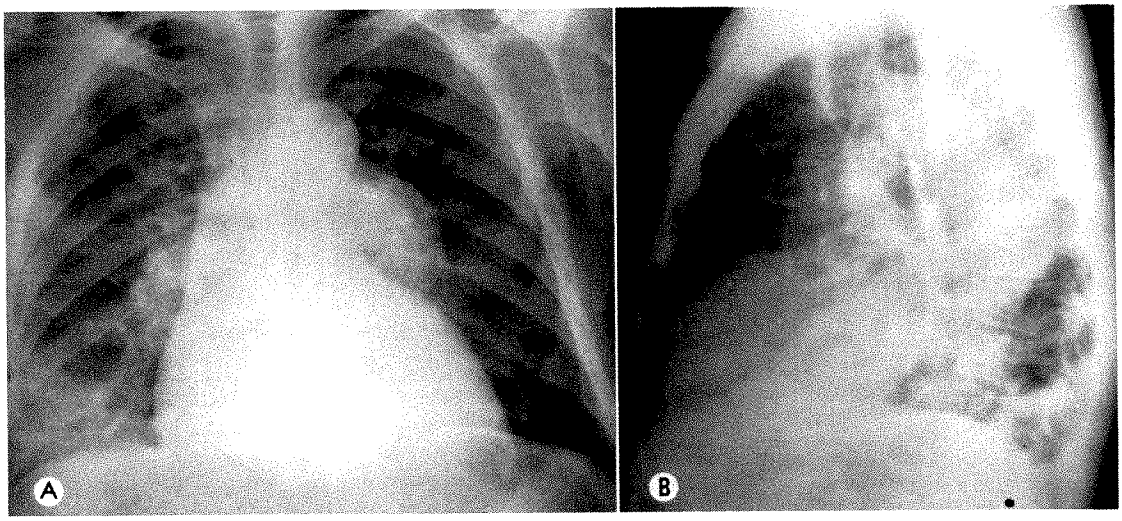

Fig. 4. Case iv. *Saccular arteriosclerotic aneurysm of the descending aorta in a 63 year old man with pulmonary infarction and heart failure.* (*A*) Frontal teleroentgenogram showing a markedly enlarged heart, right lower lobe lung infiltrations, and a left hilar mass. (*B*) Conventional lateral teleroentgenogram disclosing cardiac and thoracic aortic enlargement. Note the bulging descending aorta.

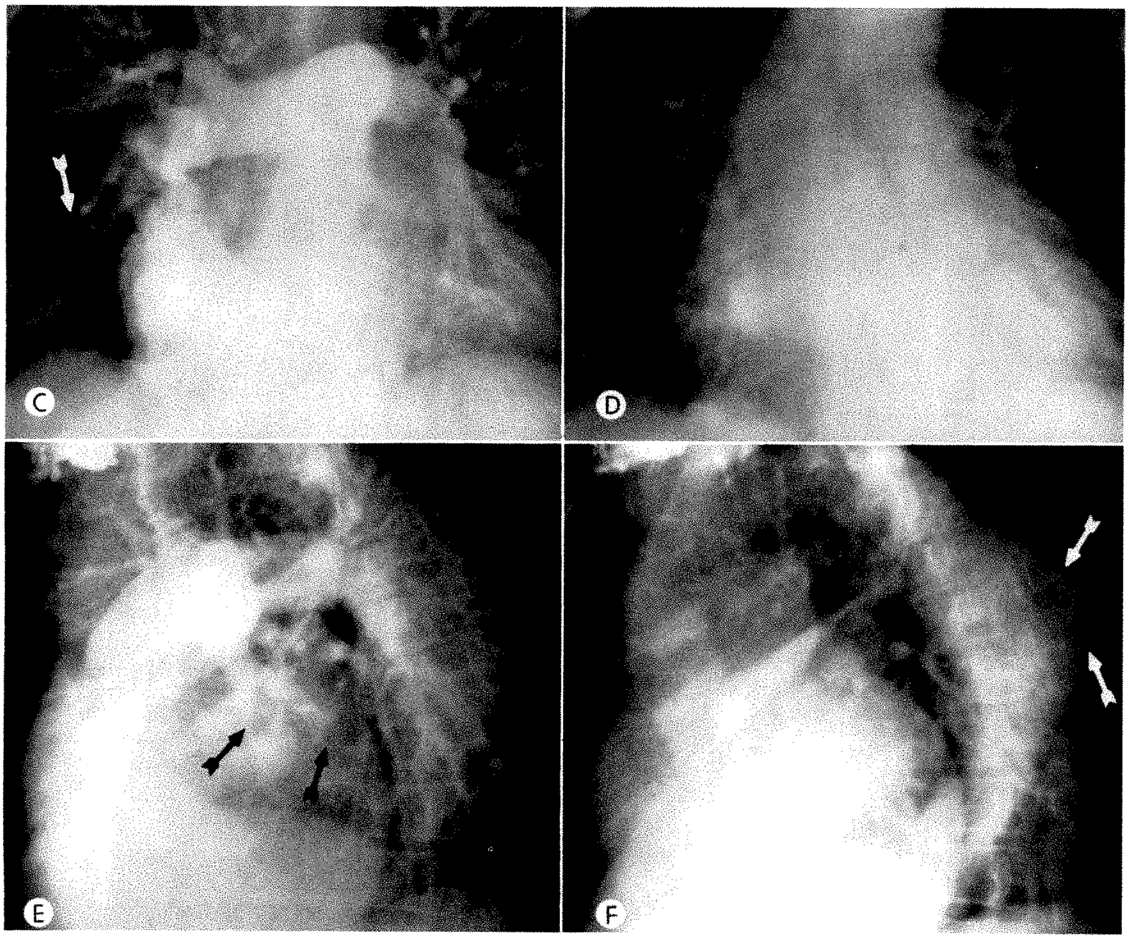

Fig. 4. (*C*) Frontal angiocardiogram at 9 seconds revealing enlargement of the right ventricle, pulmonary artery, and branches. The pulmonary arterial circulation to the right lower lobe is abruptly cut off by thrombus (arrow). (*D*) Frontal angiocardiogram later in the series at 26 seconds showing the enlarged left ventricle and dilated aorta. (*E*) Left anterior oblique angiocardiogram at 9 seconds also revealing the right ventricular and pulmonary artery enlargement. Arrows point to the occluded vessels of the descending branch of the right pulmonary artery. (*F*) Left anterior oblique angiocardiogram at 25 seconds showing the localized saccular aneurysm of the mid-outer portion of the descending aorta (arrows).

elected to perform percutaneous transfemoral aortography. A huge arteriosclerotic saccular aneurysm of the descending aorta was easily visualized (Fig. 5, *B* and *C*).

DISCUSSION

Although roentgenoscopy and roentgenography are valuable procedures in the diagnosis of aortic aneurysms, laminated clot lining aneurysms may prevent their pulsation.[4,10–14] Furthermore, tortuosity, unfolding, and kinking of the thoracic aorta may simulate aneurysms (Fig. 6 *A*, *B* and *C*).[15] Mediastinal tumors and pulmonary neoplasms may also be difficult to distinguish from vascular lesions.[23] For these reasons, contrast studies of patients with mediastinal masses have become important.[16,23] Finally, visualization of the pathologic lesions preoperatively will very often significantly influence the surgical approach and treatment of the lesion.[12]

In diagnosis of arteriosclerotic thoracic aneurysm, unfolding, elongation, and tortuosity due to arteriosclerosis and hypertension must be differentiated (Fig. 6, *A*, *B* and *C*).[13] Enlargement of the thoracic aorta

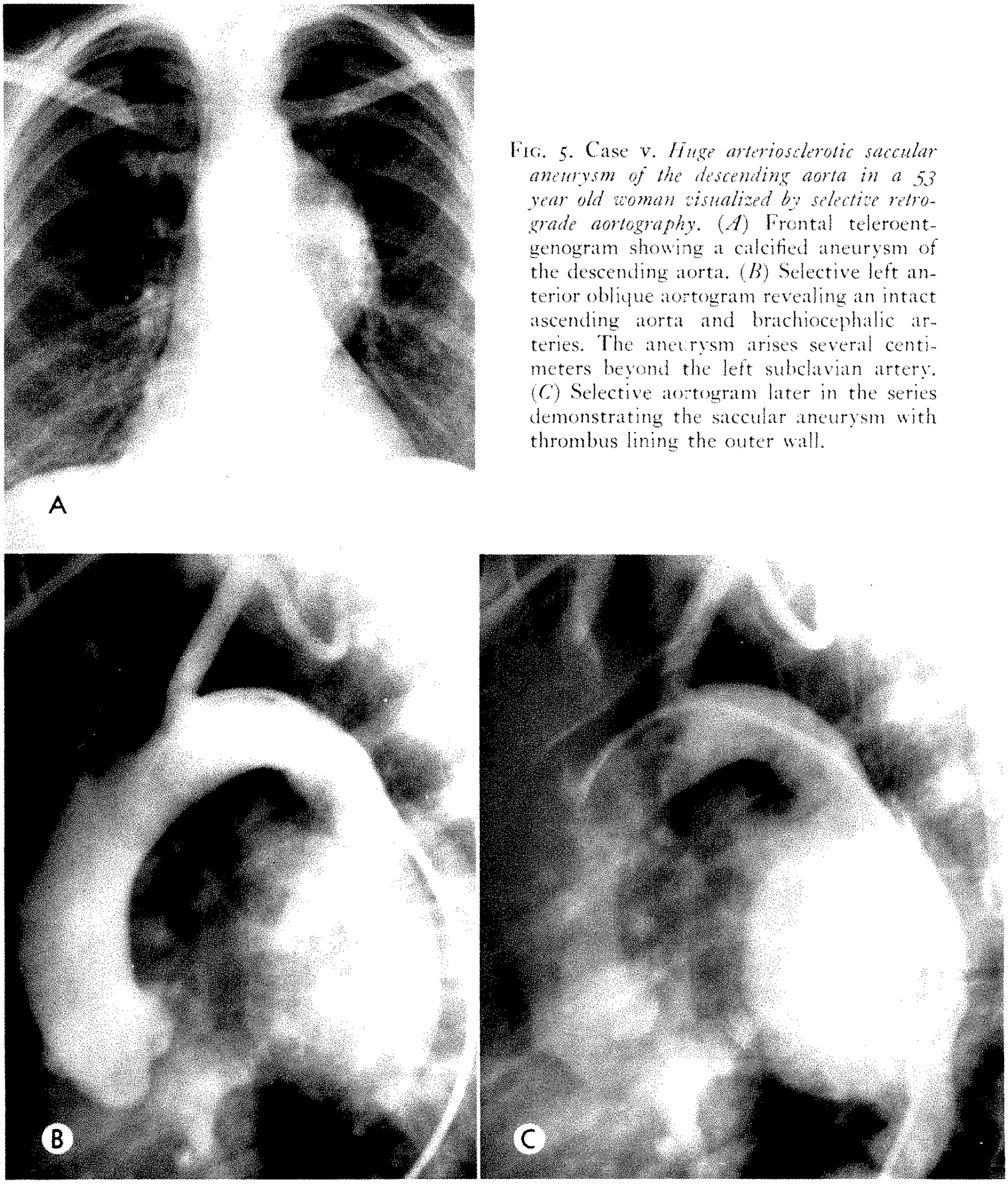

Fig. 5. Case v. *Huge arteriosclerotic saccular aneurysm of the descending aorta in a 53 year old woman visualized by selective retrograde aortography.* (*A*) Frontal teleroentgenogram showing a calcified aneurysm of the descending aorta. (*B*) Selective left anterior oblique aortogram revealing an intact ascending aorta and brachiocephalic arteries. The aneurysm arises several centimeters beyond the left subclavian artery. (*C*) Selective aortogram later in the series demonstrating the saccular aneurysm with thrombus lining the outer wall.

due to dissecting hematoma (aneurysm) must also be recognized.[1,13,15] In one chronic case, intravenous angiocardiography (Fig. 7, *A*, *B* and *C*) established the diagnosis of a dissecting aneurysm. Three acute cases of aortic dissection, visualized on percutaneous retrograde femoral arteriograms[26] and verified by operation, are shown in Figures 8*B*; 9, *A* and *B*, and 10, *A* and *B*. The last case is important because entry of the catheter into the false lumen (Fig. 10*A*) made the diagnosis difficult. Indeed, it was not until another examination was performed that the definitive diagnosis of dissecting aneurysm was established (Fig. 10*B*). The differential diagnosis of arteriosclerotic tho-

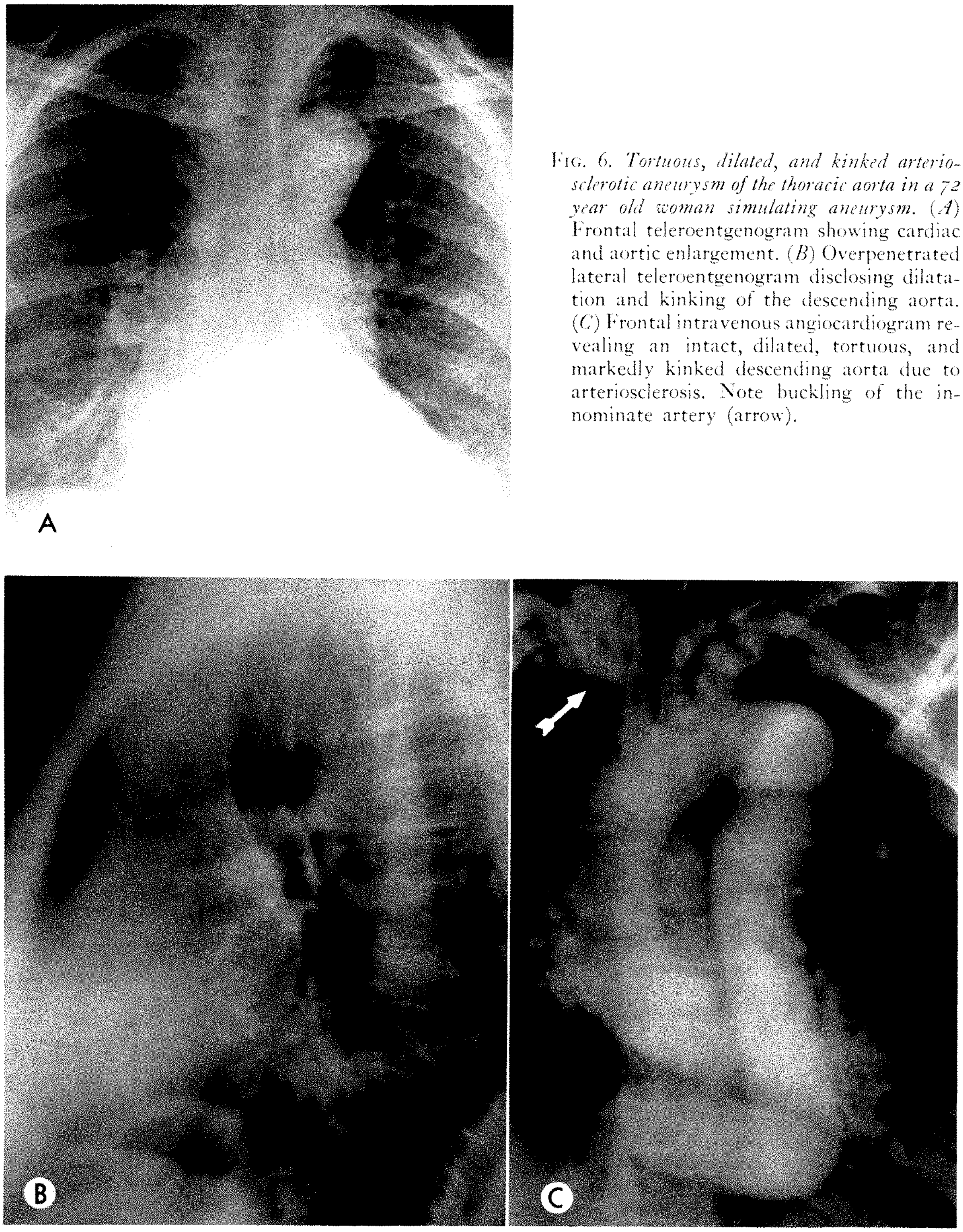

FIG. 6. *Tortuous, dilated, and kinked arteriosclerotic aneurysm of the thoracic aorta in a 72 year old woman simulating aneurysm.* (*A*) Frontal teleroentgenogram showing cardiac and aortic enlargement. (*B*) Overpenetrated lateral teleroentgenogram disclosing dilatation and kinking of the descending aorta. (*C*) Frontal intravenous angiocardiogram revealing an intact, dilated, tortuous, and markedly kinked descending aorta due to arteriosclerosis. Note buckling of the innominate artery (arrow).

racic aneurysms also includes aortic anomalies, especially of the arch, which may simulate aortic aneurysms.[22] Congenital and traumatic thoracic aneurysms may usually be differentiated from the arteriosclerotic aneurysms by the findings of the stigmata of Marfan's syndrome, or by a history of severe thoracic trauma.[16,17]

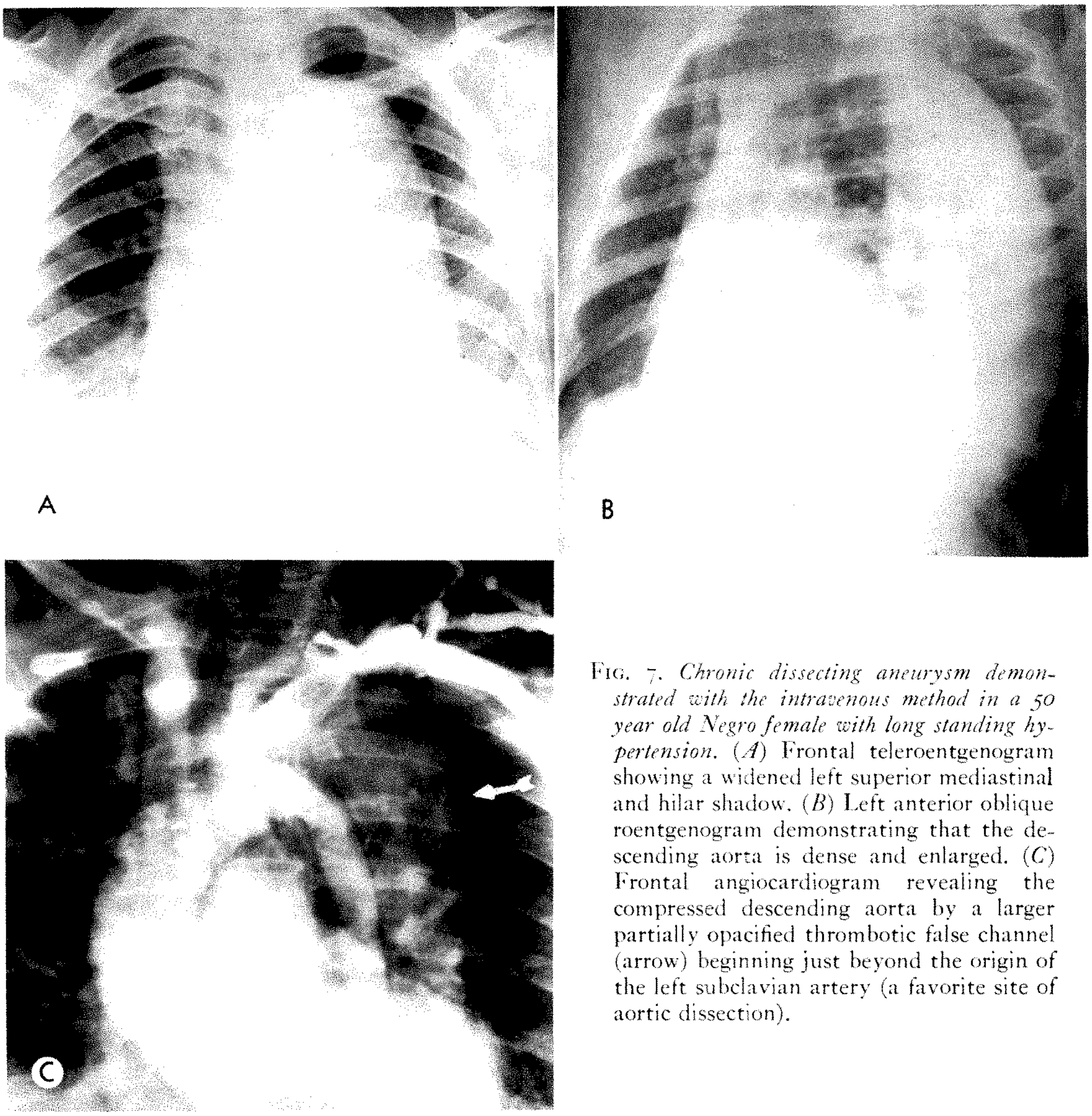

Fig. 7. *Chronic dissecting aneurysm demonstrated with the intravenous method in a 50 year old Negro female with long standing hypertension.* (*A*) Frontal teleroentgenogram showing a widened left superior mediastinal and hilar shadow. (*B*) Left anterior oblique roentgenogram demonstrating that the descending aorta is dense and enlarged. (*C*) Frontal angiocardiogram revealing the compressed descending aorta by a larger partially opacified thrombotic false channel (arrow) beginning just beyond the origin of the left subclavian artery (a favorite site of aortic dissection).

SUMMARY AND CONCLUSIONS

Arteriosclerotic aneurysms of the thoracic aorta are due to degenerative changes usually occurring in the elderly. They may produce no symptoms, but frequently there is a history of hypertension. When symptoms occur they are ominous. Backache may be due to pressure on the bodies of the thoracic vertebrae by the aneurysm; hoarseness can be caused by recurrent nerve paralysis; and hemoptysis by erosion of the pulmonary vessels by the aneurysm.

The diagnosis of aneurysms of the thoracic aorta depends upon the roentgen demonstration of mediastinal and hilar masses related to and indistinguishable from the aorta. Differentiation from mediastinal tumors may be difficult if not impossible. It then becomes necessary to perform contrast roentgenography of the cardiovascular system. This will reveal the saccular or fusiform character of aneurysms and differentiate them from arteriosclerotic dilatation, tortuosity, unfolding, and congenital anomalies of the aorta. For over 25 years intravenous angiocardiography has proved satisfactory for this. Additional information, such as the status of the renal arteries when there is associated hypertension and of the brachiocephalic arteries when cerebrovascular insufficiency is suspected, may be secured with selective aortographic techniques via the percutaneous transaxillary or transfemoral routes. The selective techniques usually provide a greater degree of radiopacity of the aorta and are even feasible, in experienced hands, when there is severe occlusive or aneurysmal disease of the iliofemoral or brachial arteries.

Intravenous angiocardiography is preferable for visualization of thoracic aneurysms when stenosis of the great veins is suspected, and when there is disease of the right heart chambers, the pulmonary artery, and of the peripheral vascular system. Ideally, both the intravenous and selective techniques should be used to enhance diagnosis and preoperative evaluation of thoracic aneurysms. In practice, however, the choice of either of the two techniques of aortography will depend upon the experience of the radiologist. Fortunately, satisfactory opacification of arteriosclerotic thoracic aneurysms can be achieved with both methods.

The diagnosis of arteriosclerotic aneurysms of the aorta depends upon exclusion of syphilis by a negative history and serologic test. However, since the treatment of an aortic aneurysm by resection of the aneurysm and replacement by a prosthesis with the aid of a bypass left atrial-femoral arterial extracorporeal apparatus is the same, whether the aneurysm is of syphilitic or arteriosclerotic etiology is only of academic interest. Arteriosclerotic aneurysms, however, are usually located in the arch and descending aorta, whereas syphilis more often involves the ascending aorta and arch.

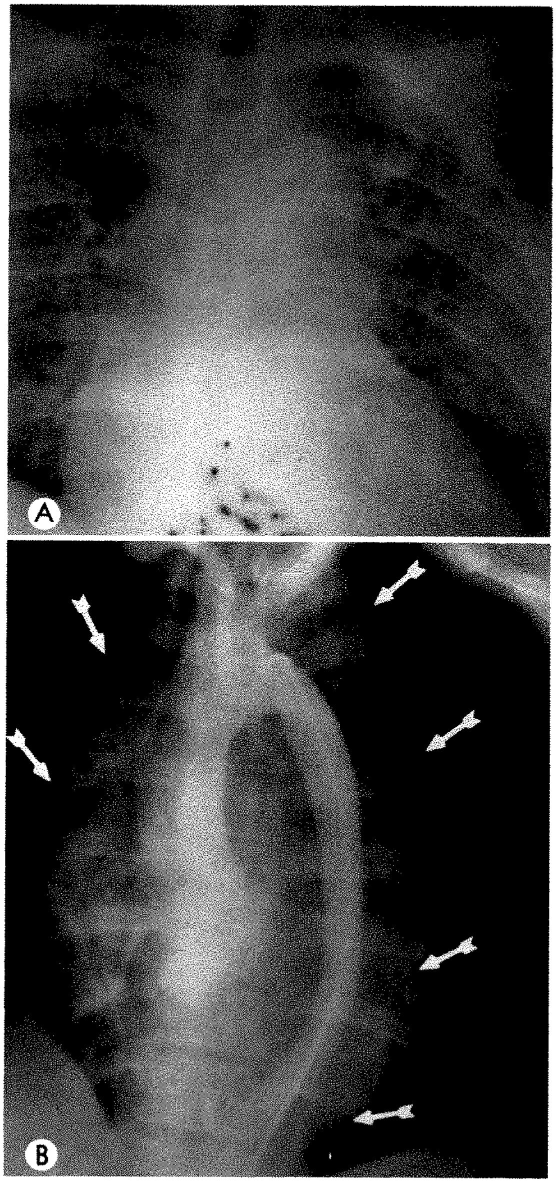

Fig. 8. *Acute dissecting aneurysm of the thoracic aorta in a 40 year old Negro with hypertension (240/120 mm. Hg).* (*A*) Frontal roentgenogram of the chest made with the mobile unit showing cardiac and aortic enlargement. (*B*) Frontal selective aortogram revealing the compressed ascending and descending aorta with deformed aortic sinuses by a false channel along the outer portions of the ascending and descending aorta (arrows). Operation though unsuccessful verified the aortographic findings.

The decision to perform surgery for arteriosclerotic thoracic aneurysms must take into account the advanced age of the patient and the tendency for arteriosclerosis to be generalized. In the heart, there may be coronary thrombosis or insufficiency; in the brain, cerebral insufficiency due to extra- or intracerebral arteriosclerosis; in the abdomen, aortic occlusive or aneurysmal disease; and in the legs, arterial occlusions or insufficiencies. Associated diseases, such as pulmonary fibrosis and emphysema, and

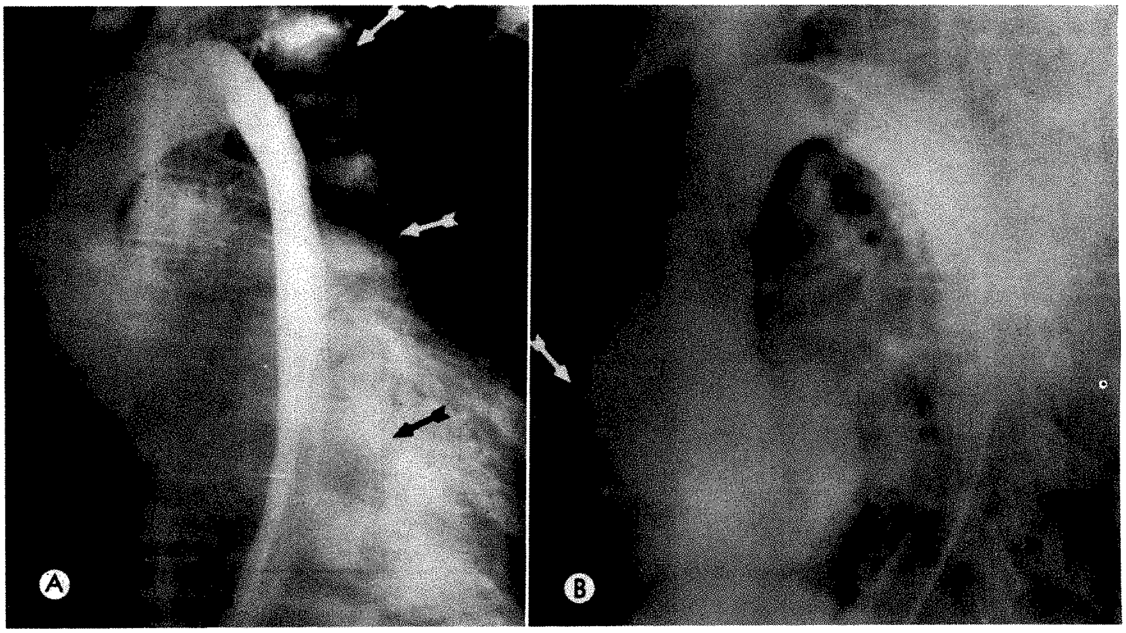

FIG. 9. *Acute dissecting aneurysm in a 47 year old Negro with hypertension (170/60 mm. Hg).* (*A*) Selective frontal aortogram with catheter introduced via the femoral route showing narrowing of the descending aorta by a large partially opacified false channel (arrows). (*B*) Another aortogram, later in the series and after the catheter was advanced towards the aortic root, revealing opacification at the aortic root and filling of a large false channel along the outer portion of the ascending aorta (arrow). Operation verified the aortographic findings. Medial cystic degeneration of the aorta was found on microscopic examination of an excised specimen of the aorta.

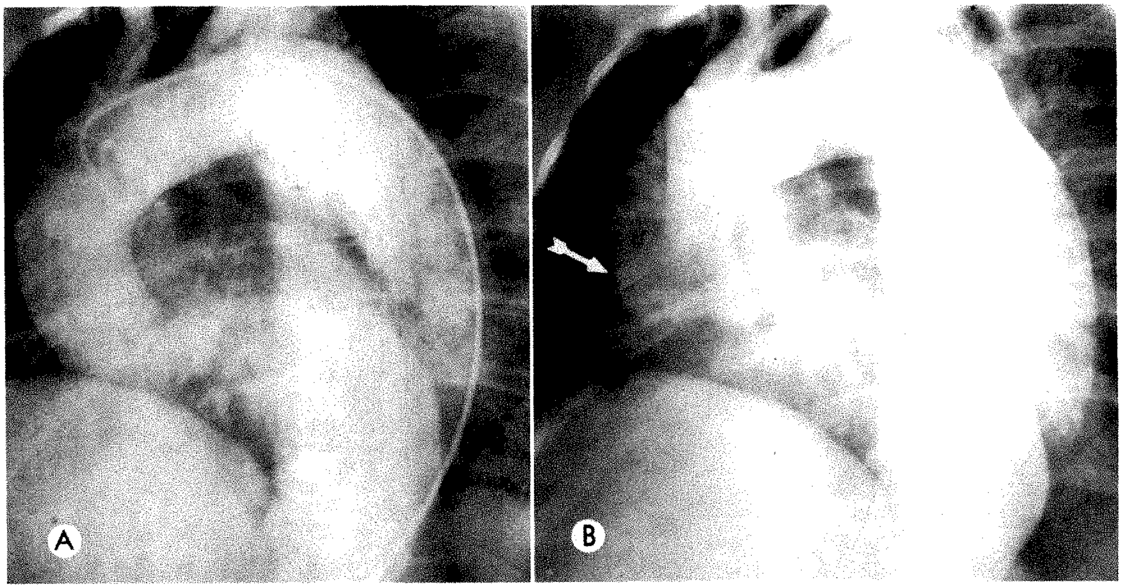

FIG. 10. *Acute dissecting aneurysm in a 55 year old Negro with hypertension (175/100 mm. Hg).* (*A*) Selective aortogram showing a widely dilated thoracic aorta. (*B*) A repeat study 5 days later revealing the classical features of aortic dissection, *i.e.*, marked narrowing and deformity of the aortic sinuses and of the descending aorta by the faintly opacified false channel (arrow). Comparison of the two studies shows that in *A* the catheter was advanced through and opacified the false channel of the aorta.

kidney or liver disease also increase the hazards of surgical resection of thoracic aneurysms. However, despite all this, the danger of rupture of the aneurysm is always present, and constant vigilance to detect the cases suitable for operation is essential.

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ORNITHOSIS

REPORT OF A CASE

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ORNITHOSIS is a viral disease of birds, called psittacosis when limited to parrots, and quite transmittable to man. In view of the difficult differential diagnosis from pneumonia, the following case is reported.

REPORT OF A CASE

A 55 year old Caucasian male physician was in excellent health prior to a routine physical examination in December, 1963, when a basal cell carcinoma, 1 cm. in diameter, was discovered on his right pinna. The diagnosis was confirmed by biopsy. He was referred to the Radiotherapy Section where he received 4,500 r to the lesion in 11 days. A chest roentgenogram at this time was negative and follow-up examinations at biweekly intervals revealed good healing of the ear without evidence of recurrence.

The patient is the Director of a research program at a large Medical Center. His past history included a tour of eastern Asia in 1962 and recent short visits to the San Joaquin Valley of California. His home is in San Francisco and he keeps no pets.

On January 27, 1964 he developed malaise, headache, myalgia and fever which progressed in a few days to such severity that his temperature remained above 102°F. His headache was not relieved by aspirin or codeine. He denied cough, dyspnea, and chest pain. The only positive physical findings on February 4, 1964 were a temperature elevation to 102°F. and a few scattered rales in the left lung. A diffuse consolidation, 4 cm. in diameter, was seen on chest roentgenograms. This lesion was located in the superior lingular segment of the left upper lobe (Fig. 1, *A* and *B*). Skin tests for coccidioidomycosis, tuberculosis, and histoplasmosis were negative. At his request, blood samples were drawn for complement fixation tests and cultures for Q fever and ornithosis. These tests were performed by his own laboratory service and were negative.

Therapy was begun with dimethylchlortetracycline hydrochloride, 600 mg. daily, vitamins and bed rest. A repeat chest roentgenogram was made on February 8, 1964 and what appeared be a more lateral involvement of the superior lingular segment was noted (Fig. 2). The patient's symptoms began to disappear and by February 13, his chest roentgenogram was almost normal (Fig. 3).

Another blood sample was taken, and at this time a complement fixation test for ornithosis was positive with a 1:64 titer. Subsequent mice culture of the organism was successful.

DISCUSSION

Ornithosis is an infectious disease caused by a filtrable virus, usually transmitted to man by birds. When these vectors are parrots and parakeets, the disease is called psittacosis. Less commonly, canaries, pigeons, seagulls, ducks, and turkeys have been responsible, and on several occasions man has been a proven carrier.[1,2,3,5,6] The virus is excreted in the nasal or anal discharge of the diseased bird and the contaminated dust is subsequently inhaled.

After an incubation period of 10 to 14 days, ornithosis manifests itself in one of three ways. The first is a mild form characterized by malaise, fever, myalgia, severe headache and pneumonitis. This lasts about 2 weeks. The second form is more severe and is referred to as the "typhoid state." The symptoms are those of the mild form, and last 4 weeks. Added to the clinical picture are abdominal pain, diarrhea, and often a spotty, erythematous cutaneous eruption not unlike the rash of typhoid fever.[5] The third form is most malignant, has a mortality rate as high as 40 per cent and may show widespread pneumonia, pleural effusion, polyarthritis, lymphaden-

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This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any indorsement of the opinions advanced or any recommendation of such products as may be named.

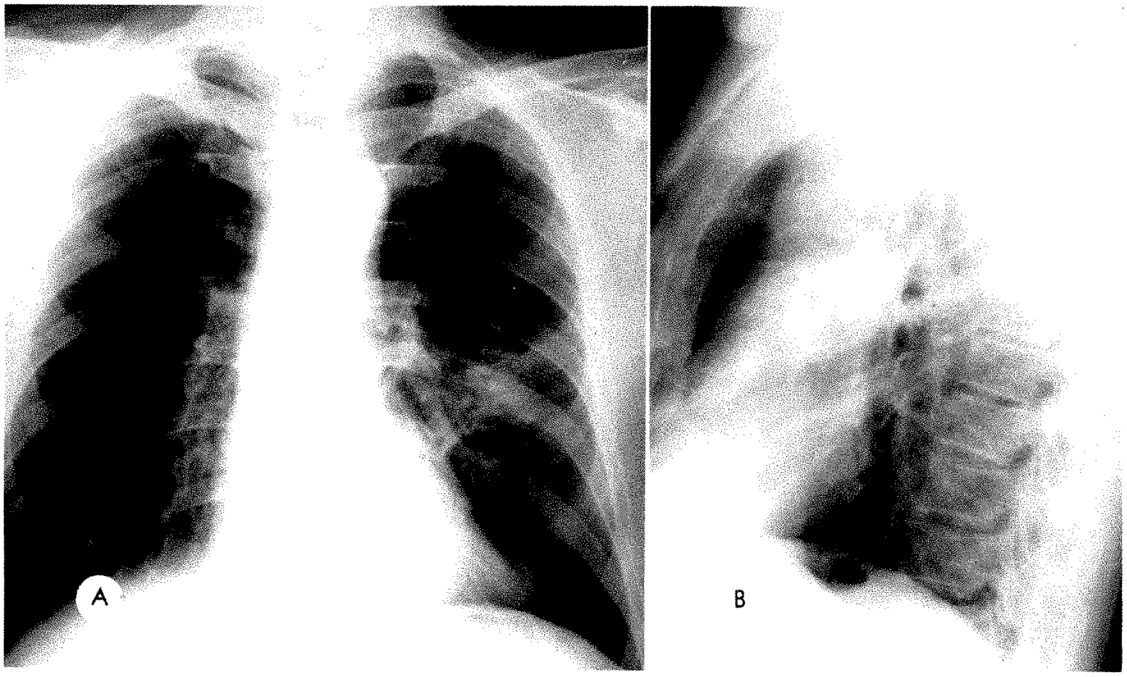

Fig. 1. (*A* and *B*) Feb. 4, 1964. Posteroanterior and lateral chest roentgenograms show a consolidation in the superior lingular segment of the left upper lobe.

opathy, splenomegaly, delirium and unconsciousness.[4,6]

A positive diagnosis is dependent upon the rising titers of complement fixation tests and the results of cultures of the virus, although a history of association with ailing birds is a helpful clue.[1,2,6] There is no

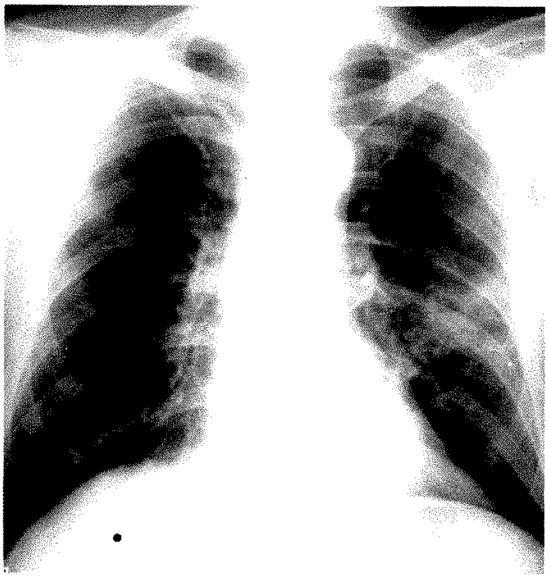

Fig. 2. Feb. 8, 1964. Posteroanterior chest roentgenogram shows a more lateral migration of the consolidation.

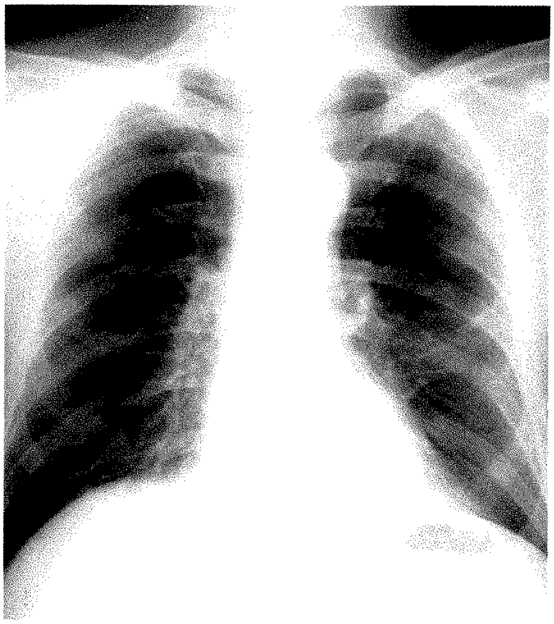

Fig. 3. Feb. 13, 1964. Posteroanterior chest roentgenogram shows almost completely resolved consolidation.

diagnostic roentgenographic picture.[2,6,7] Most commonly, chest roentgenograms show a diffuse, often striated consolidation in a lung segment which has been described as arising centrally and migrating laterally as the disease progresses, even crossing interlobar borders.[6,7] The amount of parenchymal involvement often parallels the severity of the symptoms.

Prognosis in the less advanced cases is good, and relief from symptoms is hastened by early institution of penicillin or tetracycline therapy,[3] which should be maintained for a full 14 day course to lessen the chance of recurrence.

SUMMARY

From the sparsity of cases of ornithosis reported in the English language medical literature, it is evident that the disease is infrequently diagnosed. Awareness of this entity, its communicability and clinical course, and subsequent positive serologic studies will probably increase the number of cases detected.

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SIMULATED INFILTRATION OF THE LUNG DUE TO PLEURAL THICKENING

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ON SEVERAL occasions the senior author has been called upon by members of the Chest Service to evaluate an obscure pulmonary density. Upon careful observation of the roentgen findings in posteroanterior, oblique and lateral roentgenograms along with bronchograms, it has been possible to recognize a rather typical pattern of roentgen findings in the lung attributable to localized pleural thickening. The findings may not be recognizable as such in the frontal roentgenogram, but when all of the findings are taken together it is thought that they are sufficiently characteristic to make an unequivocal diagnosis and thus to obviate the need for surgical exploration of the thorax. No discussion of the usual roentgen findings of pleural thickening is needed since they are well recognized by radiologists and chest physicians generally. On the other hand, when the findings are atypical and the pleural thickening is localized along the lateral chest wall rather than at the base they require more detailed scrutiny and analysis.

ROENTGEN FINDINGS

On the plain roentgenogram there is generally speaking an apparent infiltration in the lower lung field on one side. There is a characteristic approximation of lung markings toward the periphery of the lung adjacent to the pleural surface. There is fan-like radiation terminating near the chest wall. . . . With proper oblique and lateral roentgenograms, it will be seen that where the radiating markings terminate at or near the chest wall there is a local zone of pleural thickening which generally does not extend down to the costrophrenic angle in an obvious pattern. There is a plaque of pleural thickening, broadest in its midportion and tapering at both edges to blend with the invisible pleural line above and below.

Extensive pleural thickening may be present but not obvious. There are subtle changes of pleural thickening which should be looked for. There may be approximation of the ribs in a constant pattern which does not change with deep inspiration or expiration. There may be faint pleural calcification seen tangentially in the correct oblique projection. On rare occasions the ribs will be broadened and denser than usual due to bony proliferation. Characteristically, while there is marked approximation of lung markings, there is no real exudative or nodular infiltration when the apparent infiltration is examined in detail. The roentgen findings described are perhaps sufficiently clearcut in themselves to allow a diagnosis but if any doubt exists then bronchography should be done to clinch the diagnosis.

BRONCHOGRAPHIC FINDINGS

When the area in question is filled, the terminal bronchioles fill far out toward the periphery and radiate in a characteristic fashion toward the zone of localized pleural thickening. The bronchi can be seen to spread out from the hilus and as they extend peripherally they appear to gather up in a bundle and pucker together as one could imagine would occur if the peripheral portion of the lung were tied with a purse string.

These findings are what one might expect with scar formation as it occurs anywhere else in the body with late scarring and contraction of the pleura. The lung, which is fixed in position or "trapped" as it

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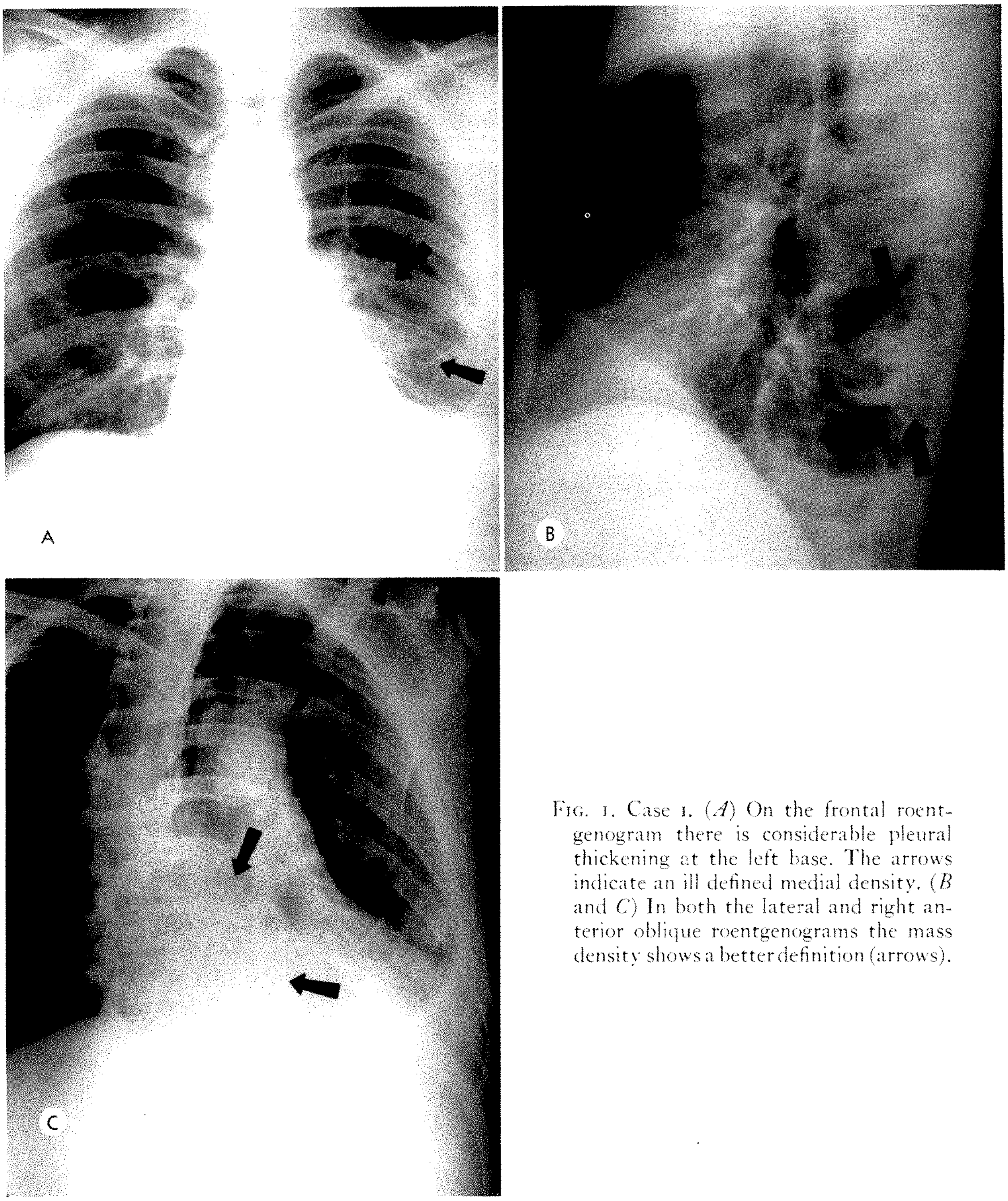

Fig. 1. Case 1. (*A*) On the frontal roentgenogram there is considerable pleural thickening at the left base. The arrows indicate an ill defined medial density. (*B* and *C*) In both the lateral and right anterior oblique roentgenograms the mass density shows a better definition (arrows).

were, must contract along with the thickened pleura which is so intimately adherent to it.

REPORT OF CASES

Case 1. A white male, aged 47, developed chest pain and cough in 1961. A survey roentgenogram with the mobile unit was interpreted as a mass in the left lower lobe. He presented himself to the Veterans Administration Hospital as a well developed man in no distress but on examination had dullness to percussion with absent vocal fremitus and diminished breath sounds in the left lower lobe. Laboratory studies, sputum cultures and pleural biopsy were negative. Thoracentesis disclosed straw colored fluid. Roentgen studies were interpreted as a mass in the left lower lobe, possibly carcinoma. A thoracotomy was done which showed a thick peel of visceral pleura involving the left lower lobe and the lingula of the left upper lobe. A segmental resection of the superior segment of the left lower lobe and decortication of the remainder of the left lower lobe were done. The lung was then seen to expand. No tumor was found. The pathologic report was: fibrous pleuritis (Fig. 1, *A–E*).

Case II. A white male, aged 30, while in the Army in 1956 developed chest pain and possible pneumonia. A follow-up chest roentgenogram at that time was read as fibrosis of the left midlung field. A routine chest roentgenogram in 1961 was interpreted as a mass in the left lung. He was admitted to the Veterans Administration Hospital. He had no complaints and his examination and laboratory studies were negative. A bronchoscopy showed no abnormality. Roentgen studies were interpreted as fibrotic changes and loss of lung volume. No change was noted in the follow-up roentgenogram 1 year later (Fig. 2, *A–D*).

Case III. A white male, aged 69, presented himself with a history of cough and hemoptysis of 1 month's duration. His physical examination, laboratory studies and bronchoscopy were negative. The roentgenogram was reported as showing a mass in the left lower lobe, posterior to the heart. Later a bronchographic study determined this to be fibrosis and loss of lung volume (Fig. 3, *A–D*).

Case IV. A white male, aged 65, entered the hospital with a history of a cough,hemoptysis and malaise of 2 months' duration. Physical examination was negative except for dullness in the right lower chest. Laboratory values were within normal limits. Pleural biopsy indicted a pleuritis. An exploratory operation was planned but the patient refused. Figure 4, *A–C* shows the roentgenographic findings in this patient.

Case V. A white male, aged 41, entered the hospital after having two severe upper respiratory tract infections in 3 months. He had also lost weight and developed clubbing of the fingers. Physical, laboratory, and biopsy examinations were all negative. The roentgen diagnosis was possible tumor of the upper lobe of the left lung. Operative results were negative for

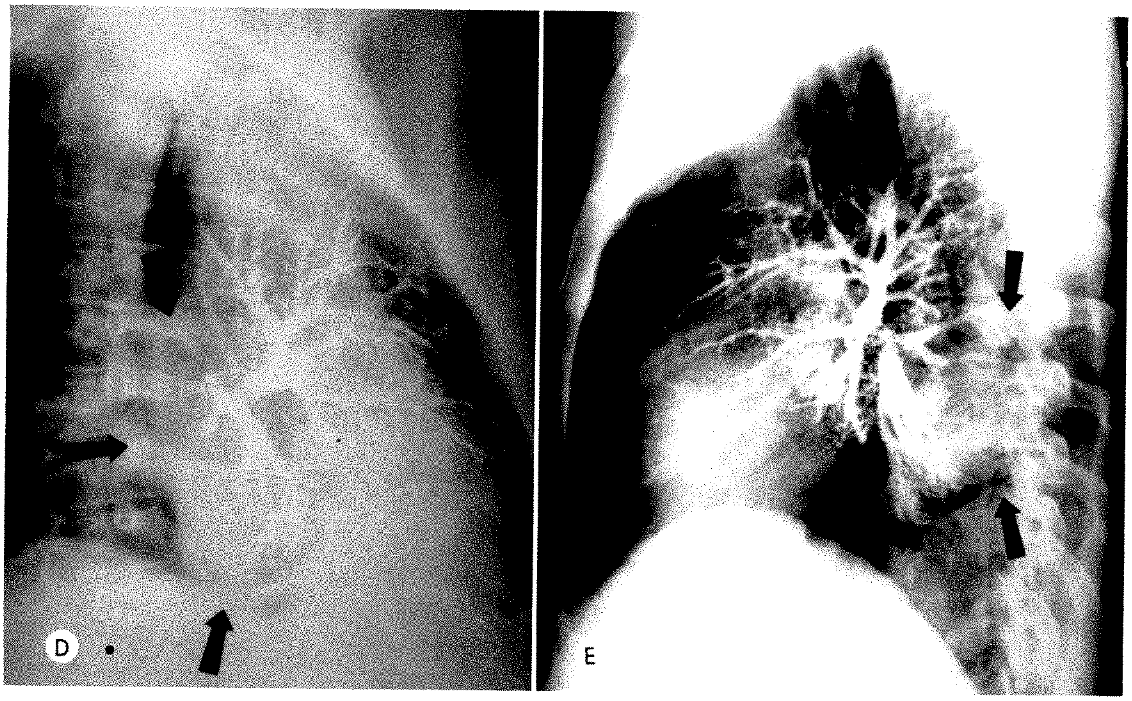

Fig. 1. (*D* and *E*) Left bronchograms. In the oblique and lateral projections, the bronchi are only partially filled in the area of the mass. These approximated bronchi converge toward the pulmonary density and occupy less than the usual volume.

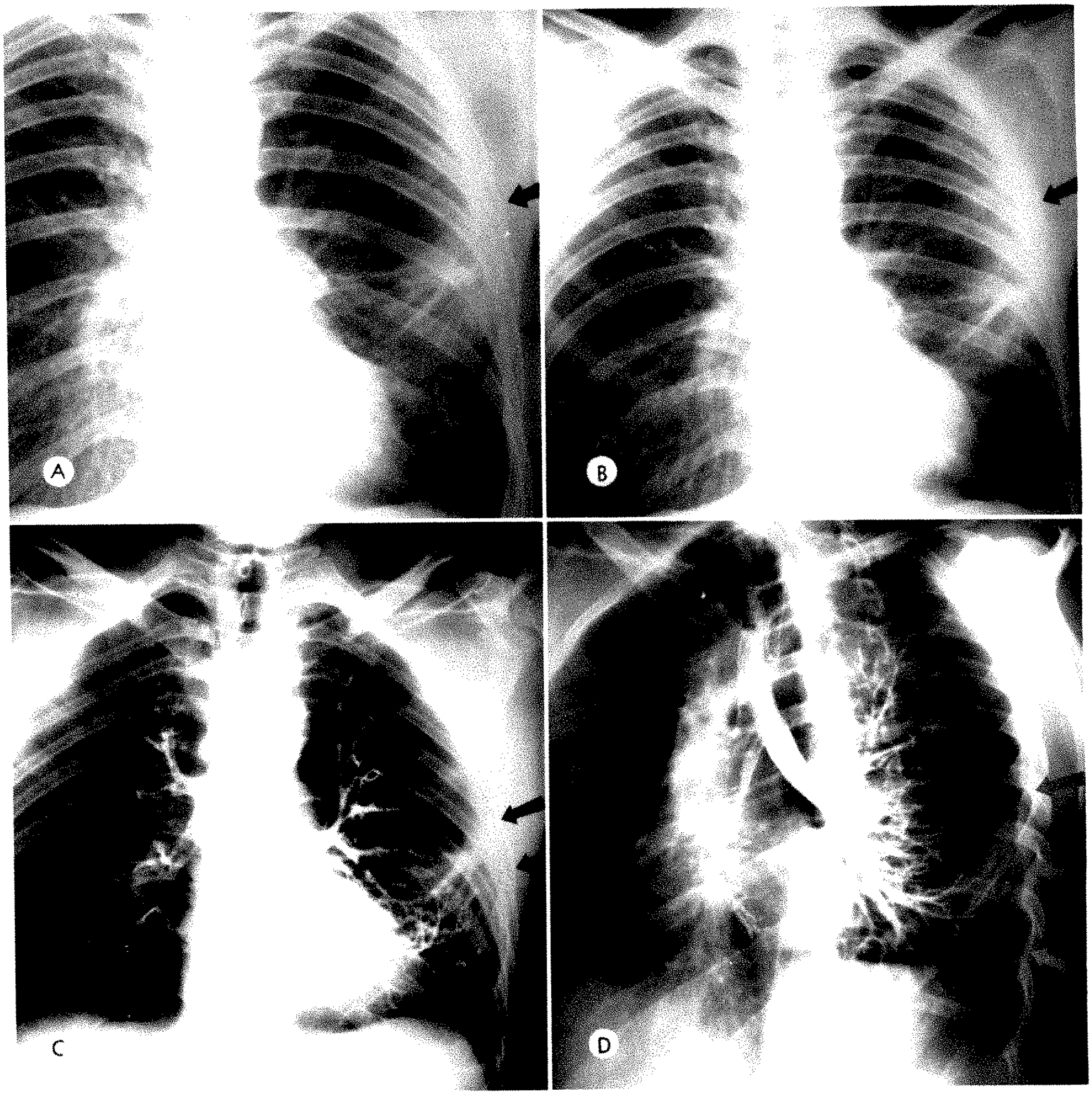

FIG. 2. Case II. (*A* and *B*) No change is noted in the posteroanterior roentgenograms of 1961 and 1962. Note the thickened pleura toward which there is puckering of the pulmonary infiltrate. (*C* and *D*) Left bronchograms. In the posteroanterior and left anterior oblique projections there is radiation of the bronchi toward the plaque-like pleural thickening (arrows).

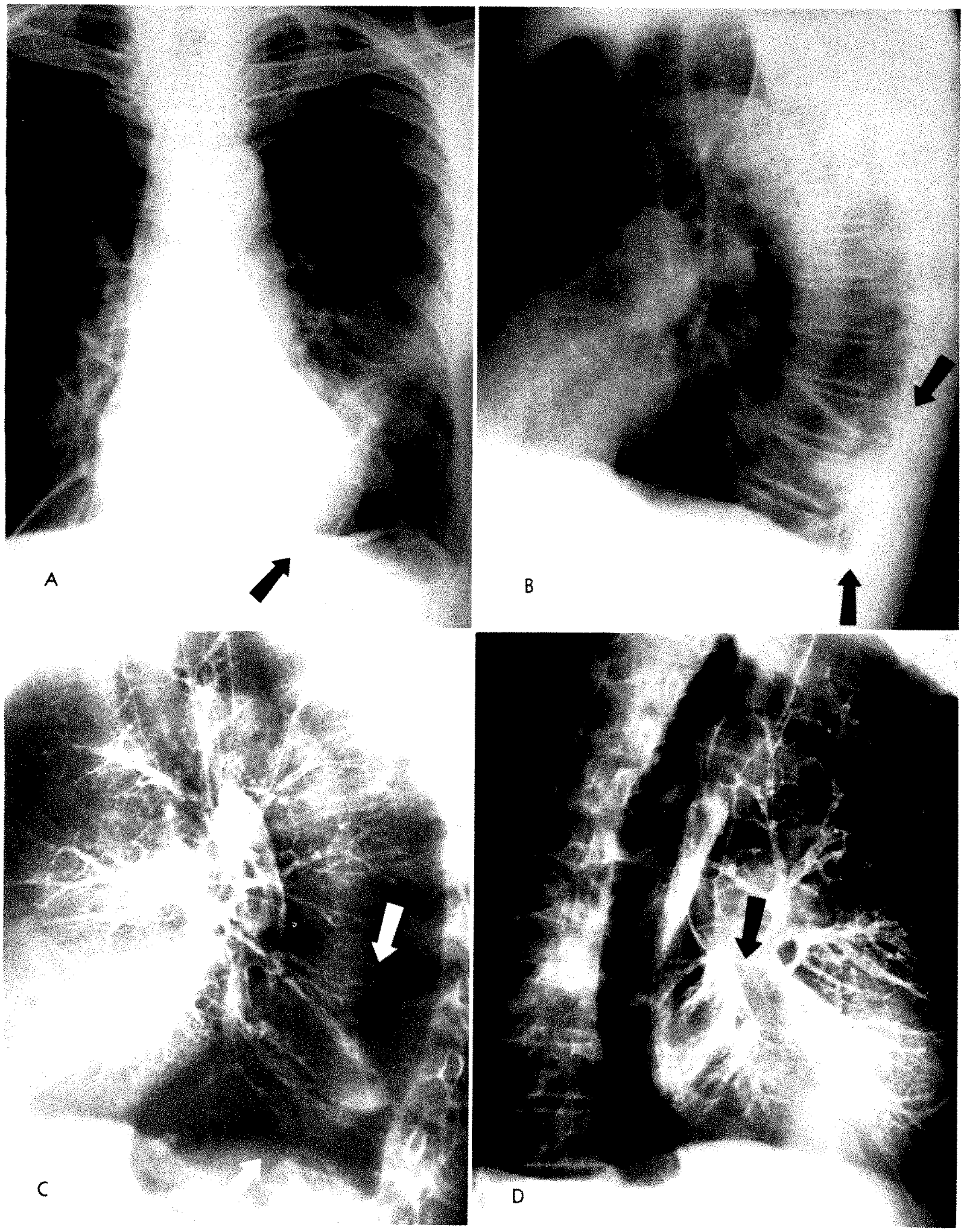

FIG. 3. Case III. (*A*) On the posteroanterior roentgenogram there is seen an ill defined retrocardiac density that extends above the cardiac apex. (*B*) The arrows on the lateral roentgenogram indicate the upper and lower limits of the parenchymal density. (*C* and *D*) Left bronchograms. Note the windswept appearance of the radiating bronchi. The peripheral approximation of the bronchi (arrows) toward the plaque of pleural thickening, posteriorly, is in sharp contrast with the spreading of the bronchi in the upper lung field.

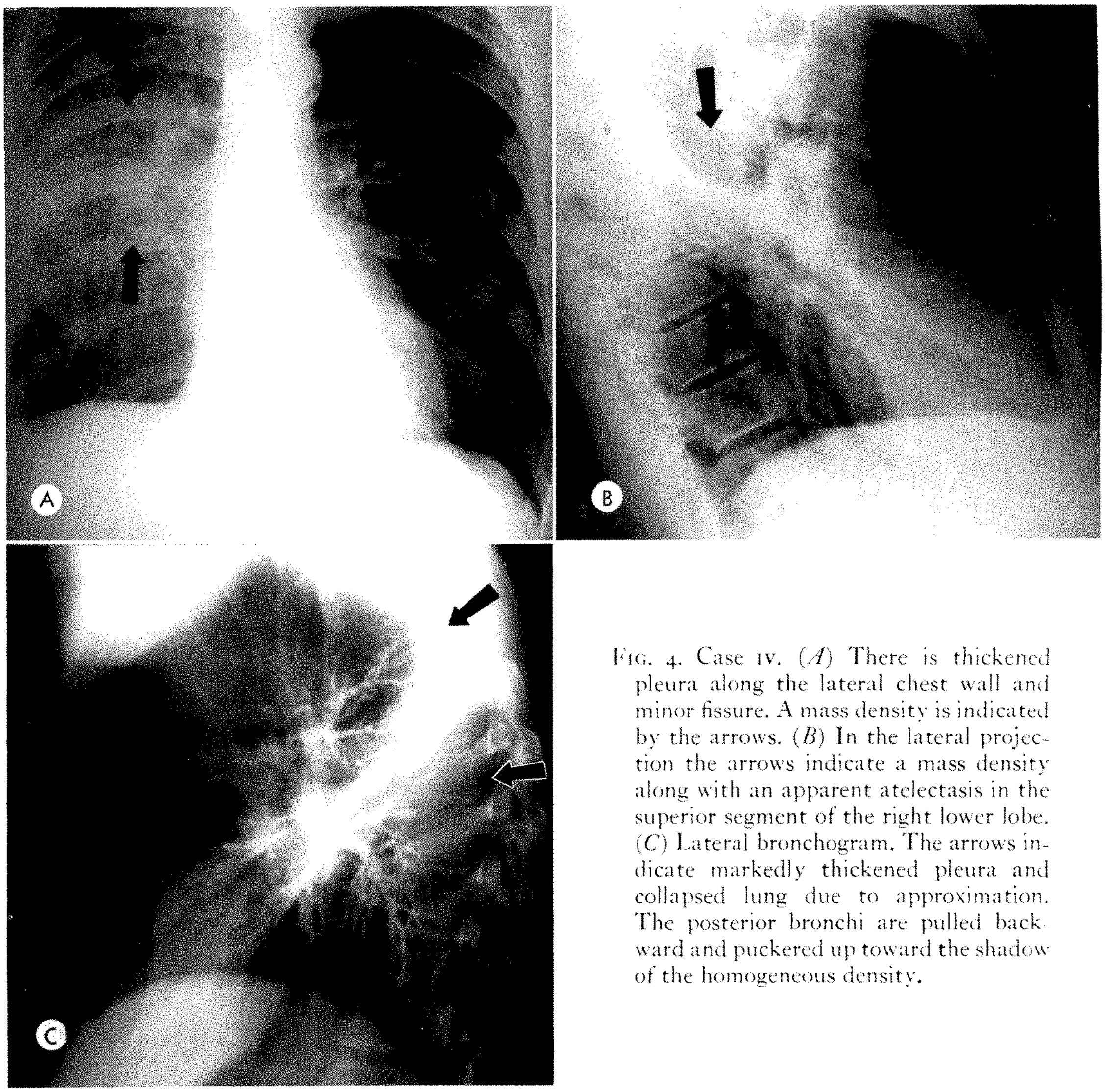

FIG. 4. Case IV. (*A*) There is thickened pleura along the lateral chest wall and minor fissure. A mass density is indicated by the arrows. (*B*) In the lateral projection the arrows indicate a mass density along with an apparent atelectasis in the superior segment of the right lower lobe. (*C*) Lateral bronchogram. The arrows indicate markedly thickened pleura and collapsed lung due to approximation. The posterior bronchi are pulled backward and puckered up toward the shadow of the homogeneous density.

tumor but a thick retracted area of pleura enclosed the superior lingular segment of the left upper lobe (Fig. 5, *A–D*).

SUMMARY AND CONCLUSIONS

A cause of obscure pulmonary density which in reality is not an infiltration or tumor but simulates these entities very closely is discussed. It is felt that with knowledge of the roentgen findings and proper study of the patient roentgenologically an accurate diagnosis can be made and surgery or exploration avoided. Most of these patients are asymptomatic, but do have a history of previous chest pathology which usually is obtained in retrospect.

When the plain roentgen findings in multiple projections of the chest are combined with those of bronchography, a characteristic pattern evolves. This is quite pathognomonic of localized pleural thickening with trapping and segmental loss of volume of lung.

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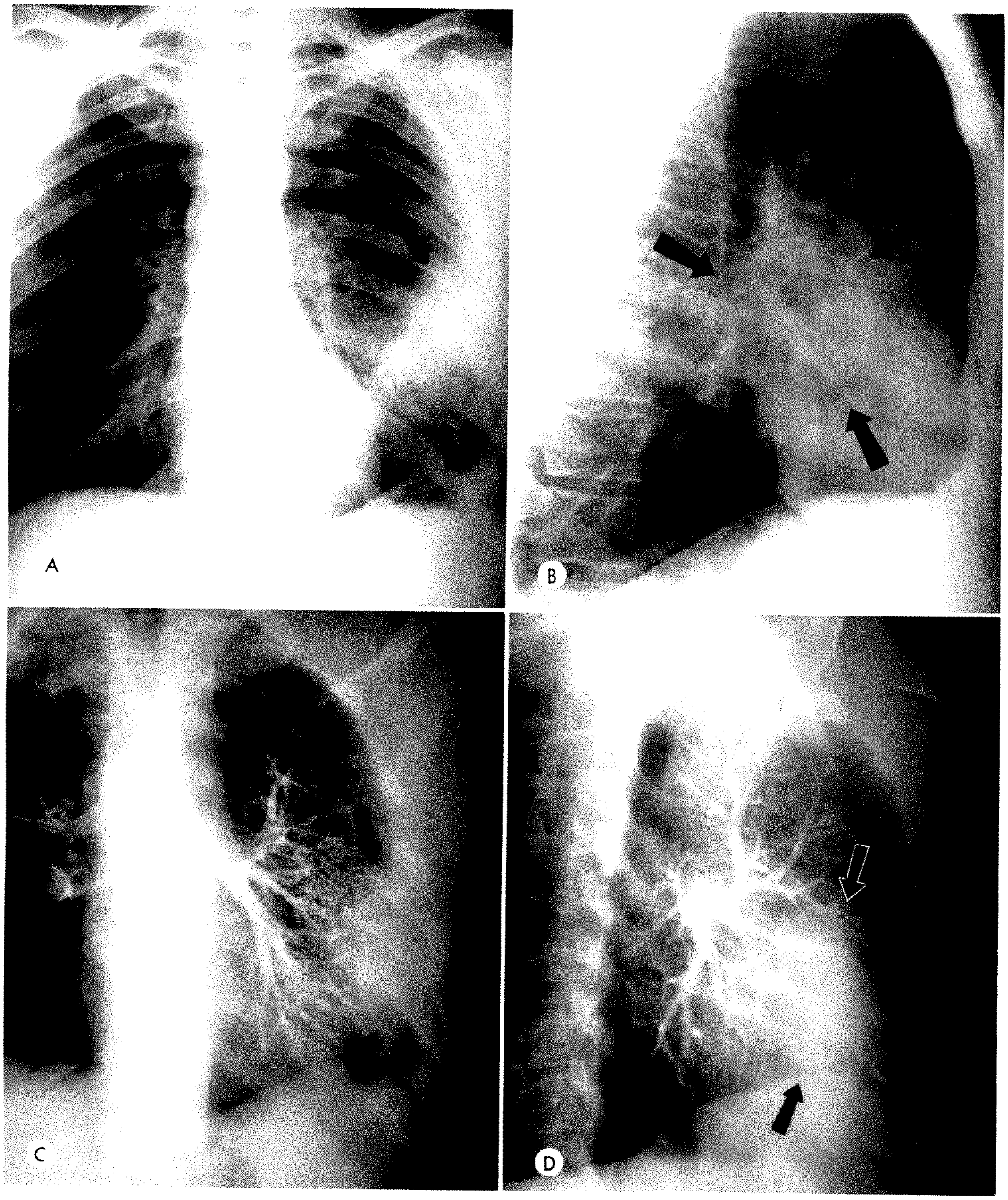

Fig. 5. Case v. (*A*) Posteroanterior roentgenogram. A wedge shaped shadow of increased density is indicated by the arrows. Some evidence of thickening of the pleura is also present. (*B*) Lateral roentgenogram. A mass density overlaps the hilus. (*C* and *D*) The posteroanterior and right anterior oblique bronchograms show marked approximation of the bronchi with radiation of these bronchi toward the zone of pleural scarring.

SOME NORMAL RENAL VARIANTS*

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BECAUSE of the inadequacy of classic intravenous pyelography in detecting renal vascular hypertension, the authors have been employing a technique of rapid intravenous injection of large volumes (1 ml./kg. of body weight) of a concentrated diatrizoate compound, immediately followed by multisection nephrotomography. A number of findings not hitherto appreciated were encountered in interpreting these pyelograms. In an effort to elucidate these problems, the authors reviewed 200 such pyelograms in addition to autopsy specimens of kidneys (*in situ* and after removal from the body). The purpose of this paper is (1) to discuss the normal variants found in this study which may be confused with renal mass lesions and (2) to point out some additional interesting observations.

I. SUPRAHILAR BULGE

Early in the course of the study, a 67 year old man was admitted to the Hospital of St. Raphael with symptoms of prostatism and hematuria. During the nephrotomographic phase of the intravenous pyelogram, a rounded area of increased density was noted above the left renal hilus (Fig. 1). Although one of the authors (R.S.) thought that this finding was normal because he had observed it previously, a number of experienced radiologists and urologists believed that the patient had a vascular lesion, *i.e.*, tumor, aneurysm or vascular malformation. To resolve this difference of opinion, a percutaneous transaxillary aortogram and a selective left renal arteriogram were made which proved to be completely normal (Fig. 2). Following prostatectomy, his symptoms including the hematuria disappeared.

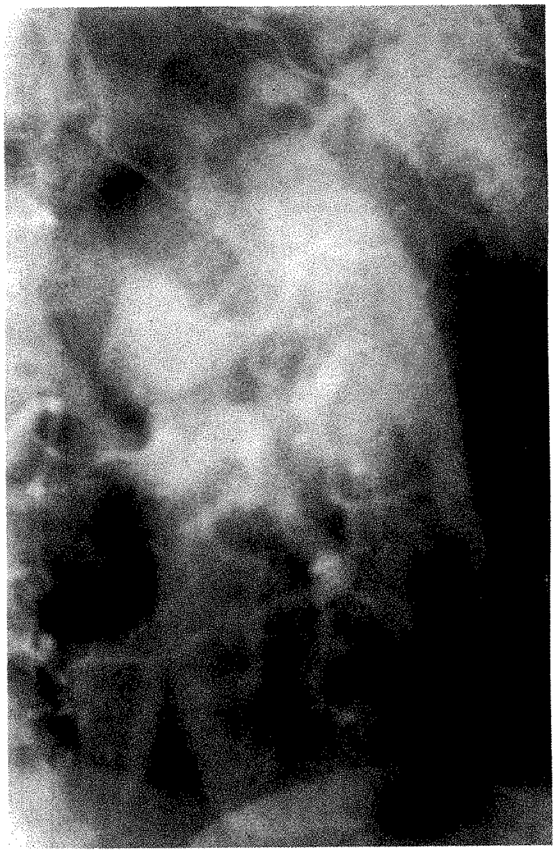

FIG. 1. Suprahilar bulge of left kidney originally visualized during the nephrotomographic phase of an intravenous pyelogram. The bulge is more dramatically emphasized on this early nephrogram taken during selective left renal arteriography.

A review of the nephrotomograms in the present series showed a number of examples of a localized bulge of the renal parenchyma just above the hilus, continuous with the medial surface of the upper pole. This was more common on the left side, there being only a single example on the right side in the material studied. Because the bulge overhangs the renal sinus, it tends to give the kidney the appearance of a fetus in the fetal attitude. The bulge is often visualized as a suprahilar localized zone of increased radiopacity during the nephrographic phase of the pyelogram. Minor degrees of suprahilar bulging are not at all infrequent. However, the prominent, densely opacified bulge which may be confused with neoplasm or a vascular lesion is less common.

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None of the patients in our series who exhibited suprahilar bulging by nephrotomography has come to necropsy. Examination of the external surface of other kidneys removed at necropsy shed little light on the problem. However, when the fat investing the renal sinus was dissected away, there was frequently noted along the medial aspect of the kidney a smooth, varying-sized knuckle of cortex which bulged down into the upper pole of the renal sinus (Fig. 3 and 4). A comparable cortical overhang of similar magnitude has not been observed to date in the lower half of the renal sinus, although Olsson and Jönsson[15] in their excellent monograph briefly allude to bulges above and below the hilus as "hilar lips." Since only renal parenchymal tissue is opacified during nephrotomography and the fat within the renal sinus is not opacified, the cortical nodule stands out as a definite bulge, protruding down into the superior portion of the hilus (Fig. 5, 6, 7 and 8).

The principal problems in differential diagnosis are concerned with the exclusion of vascular abnormalities; *e.g.*, renal artery aneurysm and related malformations (Fig. 9 and 10) and neoplasm. Aneurysm and other vascular malformations can be ruled out by renal arteriography, although this procedure is hardly necessary in the typical case. Likewise, tumor is not an issue of grave concern in the presence of the following triad: (1) characteristic location and appearance of the suprahilar bulge; (2) normal upper pole collecting system; and (3) normal vasculature demonstrated by renal arteriography.

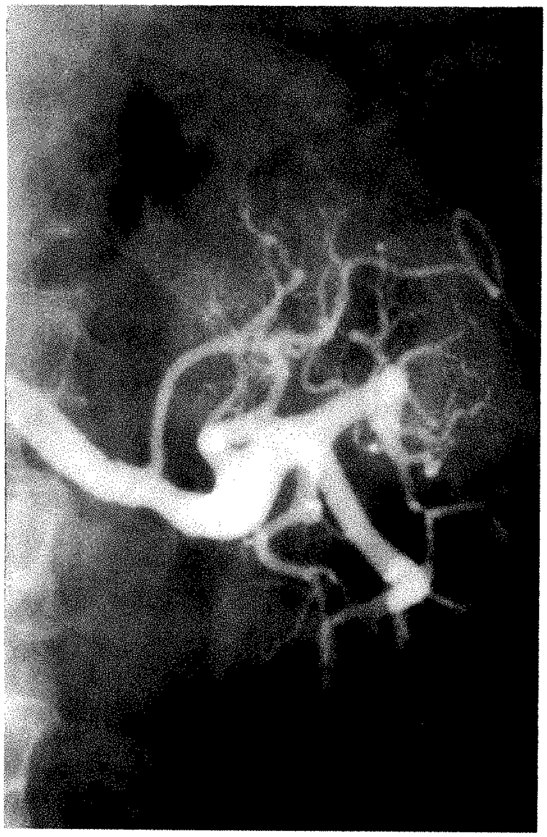

Fig. 2. Selective left renal arteriogram demonstrating normal arterial pattern.

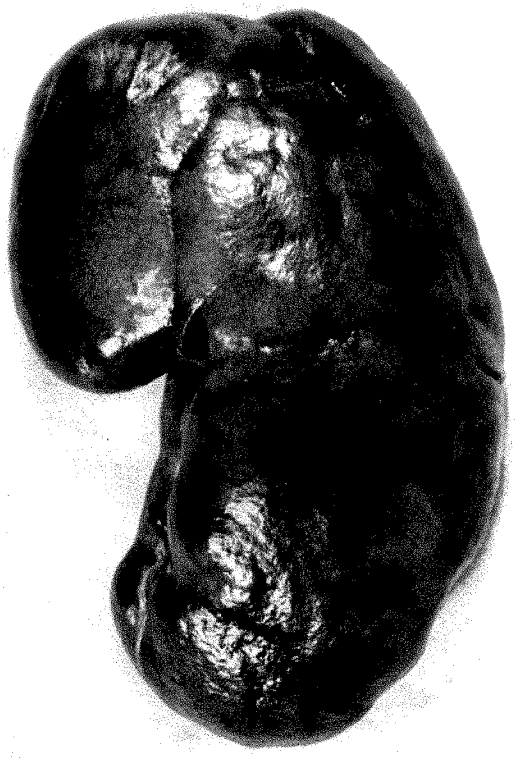

Fig. 3. Photograph of a left kidney directly *en face* in the anteroposterior position showing a prominent suprahilar bulge (necropsy specimen).

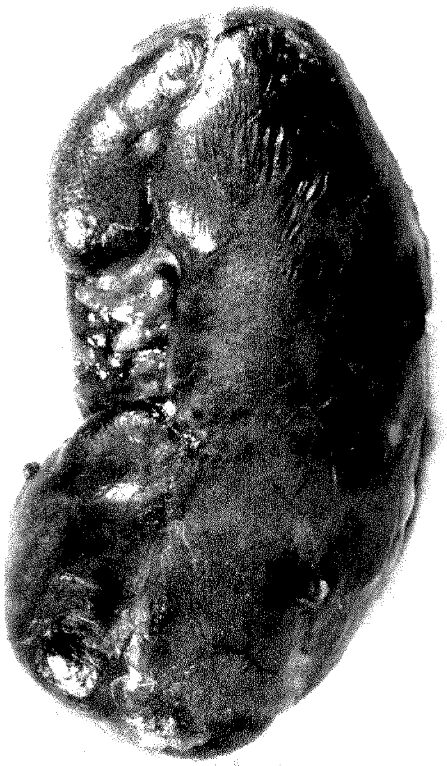

Fig. 4. Photograph of the same kidney specimen as in Figure 3 following partial removal of the fat in the hilus and slight external rotation.

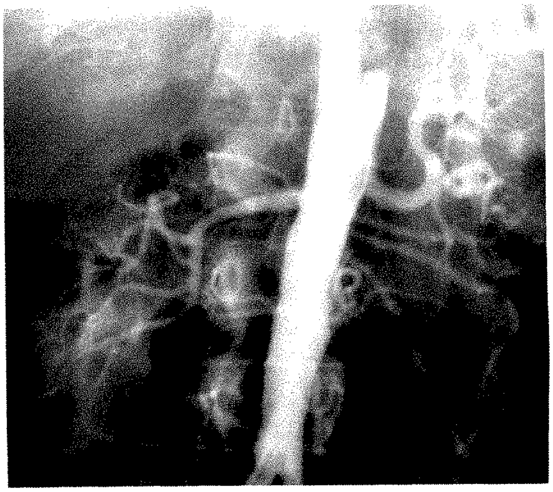

Fig. 6. Arterial phase of the aortogram of the same patient as in Figure 5 demonstrating normal renal vasculature. Two renal arteries are present on the left side.

II. THE "NORMAL" LARGE KIDNEY

Moëll[13,14] established a normal range of values for renal size after measuring a large series of roentgenograms. According to Moëll, the kidney in the male is somewhat

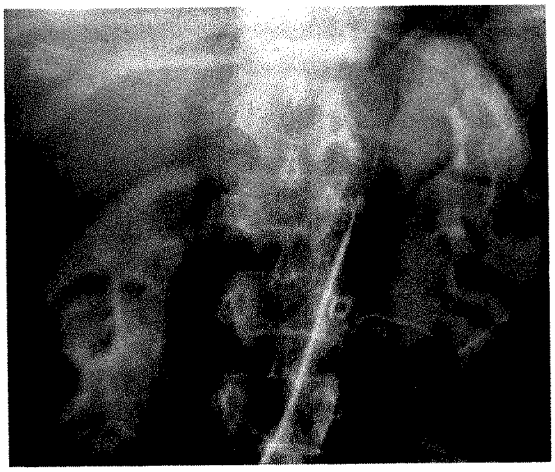

Fig. 5. Nephrographic phase of aortogram showing a suprahilar bulge on the left with a normal pelvicalyceal system. (Courtesy A. M. Diggs, M.D., Danbury, Connecticut.)

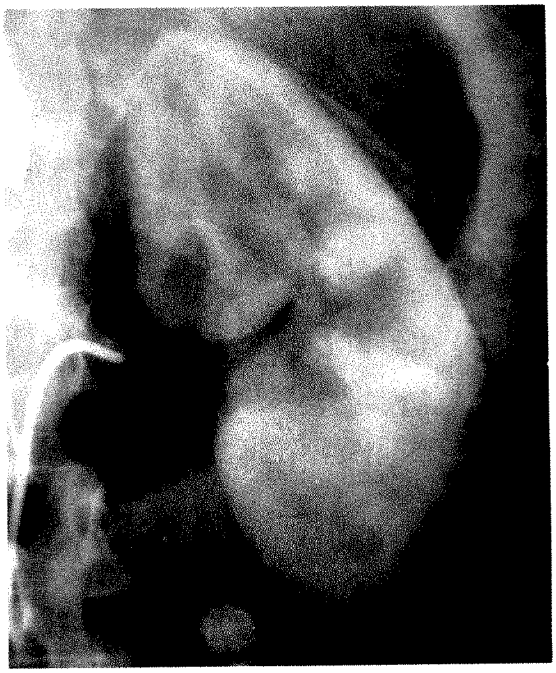

Fig. 7. Pronounced left suprahilar bulge seen during the nephrographic phase of a selective left renal arteriogram.

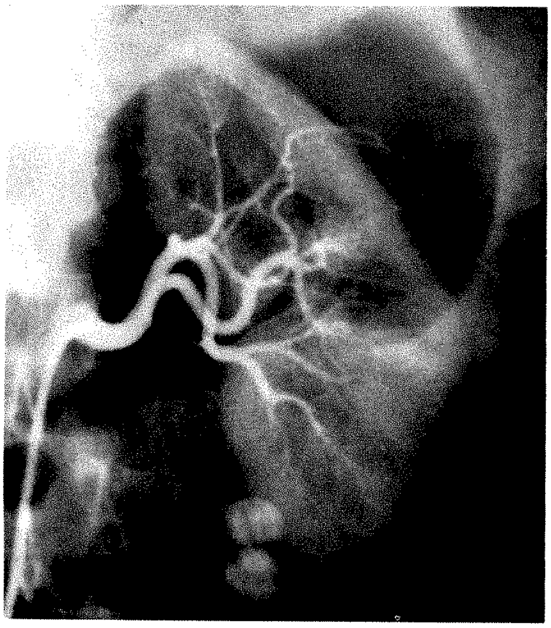

Fig. 8. Normal selective left renal arteriogram in the same patient as in Figure 7.

larger than in the female and the left kidney in both sexes is somewhat larger than the right. The average dimensions in males are 12.9 cm.×6.2 cm. for the right kidney and 13.2 cm.×6.3 cm. for the left. The corresponding measurements in females are 12.3 cm.×5.7 cm. and 12.6 cm.×5.9 cm., respectively. Most authors[9] agree that a disparity in renal size between the two kidneys greater than 1.5 cm. is significant, the abnormal kidney being smaller because of renal ischemia or other parenchymal disease.

Recently, we have encountered an example of unilateral diffuse renal enlargement and two examples of unilateral localized renal enlargement. In each of these cases, the contralateral kidney was normal in size, thereby excluding the usual type of compensatory hypertrophy.

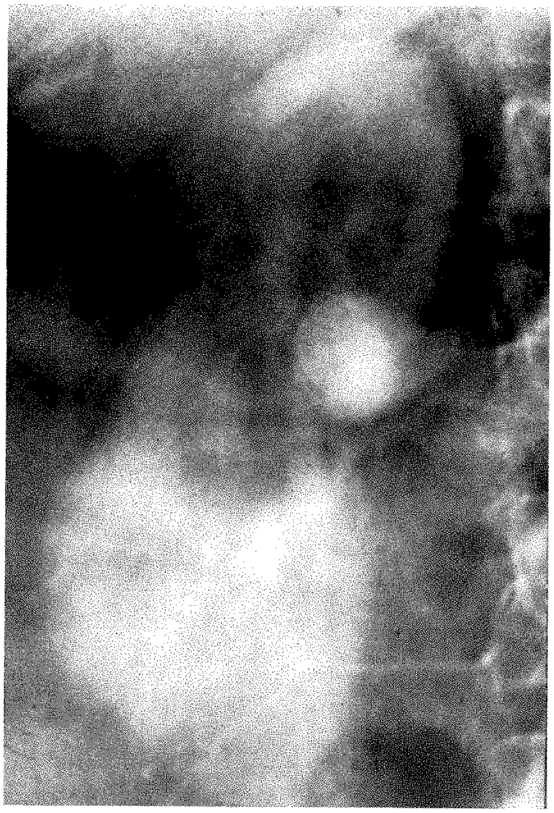

Fig. 9. Nephrotomographic phase of aortogram showing an aneurysm of the right renal artery in the region of the renal hilus.

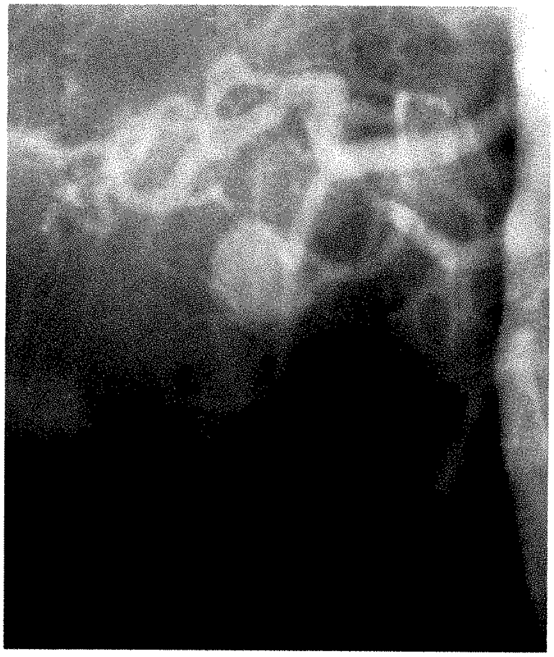

Fig. 10. Opacification of the aneurysm in the same patient as in Figure 9 during the arterial phase of the aortogram.

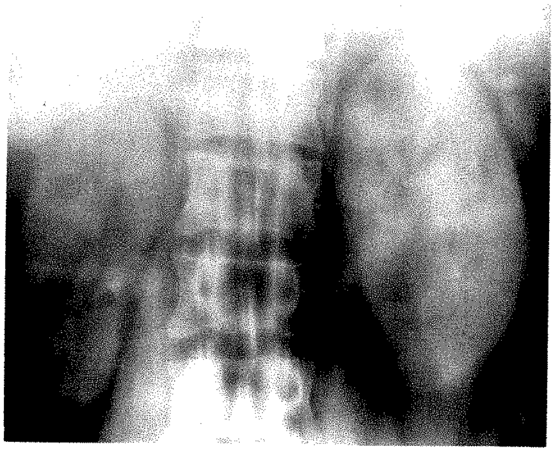

Fig. 11. Case I. Nephrotomogram showing diffuse generalized enlargement of the left kidney.

A. DIFFUSE ENLARGEMENT OF ONE KIDNEY

Case I. J.G., a 39 year old normotensive white male, had experienced several mild episodes of right renal colic during the 3 years prior to admission to the hospital. His health, otherwise, had been excellent. Two weeks before entering the hospital, he developed right flank pain and tenderness which persisted up to the time of admission. Examination of the urine including cytology was essentially negative. Conventional intravenous and retrograde pyelography revealed a normal sized right kidney (14.5 cm.) containing a 1 cm. nonobstructing calculus in the renal pelvis. The left kidney, however, was considerably enlarged measuring 17 cm. in length. Nephrotomography and selective left renal arteriography showed a symmetrically enlarged kidney with a normal calyceal system and normal vasculature (Fig. 11 and 12). There was also a suprahilar bulge present. The spleen appeared to be normal in size and location. Following removal of the calculus by right pyeloureterotomy, the patient had an uneventful postoperative course and was discharged from the hospital in good condition.

Because there was no opportunity to study the enlarged left kidney histologically, it cannot be definitely proven that the enlargement represented true hypertrophy. Indeed, hypertrophy of one kidney in the presence of a contralateral normal kidney has, to the authors' knowledge, not been described (admittedly our patient's right kidney was not entirely normal). Renal hypertrophy is usually a compensatory phenomenon which occurs in the presence of significantly decreased function, or absence, of the other kidney.[12] Thus, following unilateral nephrectomy in the rat, histologic changes can be detected in the remaining kidney as early as 4 days postoperatively with increasing hypertrophy up to approximately 6 weeks.[8] This type of renal enlargement is characterized both by hyperplasia and hypertrophy of all the cells comprising the nephron but is most prominent in the glomerulus and proximal tubules. Compensatory hypertrophy is influenced by a number of factors including age, dietary intake of protein and anterior pituitary function.[1,5,7] Jackson and Shiels[10] showed that the degree of compensatory hypertrophy in the remaining kidney of a rat following uninephrectomy is considerably greater in young rats (1 month) than in adolescent or mature rats. This observation parallels the clinical experience in man

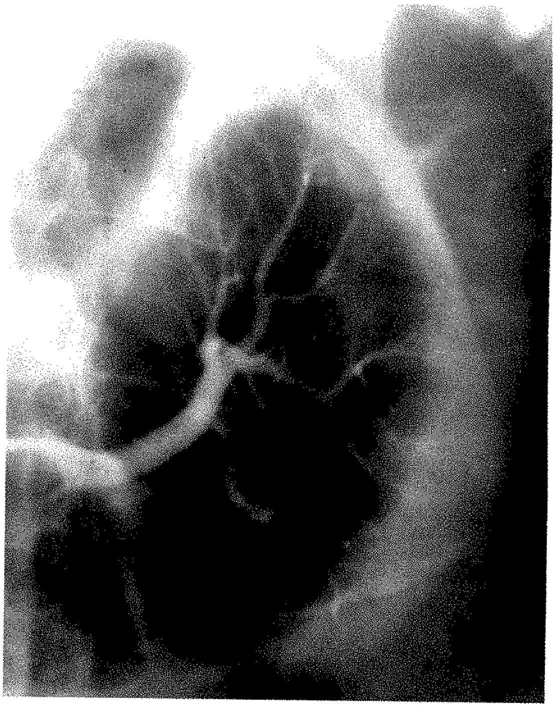

Fig. 12. Case 1. Selective left renal arteriogram demonstrating normal vasculature.

that patients younger than 30 years of age usually exhibit significant hypertrophy within a month following nephrectomy while patients above the age of 50 years show minimal hypertrophy of the remaining kidney. Adequate ingestion of protein and the administration of testosterone enhance the hypertrophic reaction by contributing to the supply of protein building blocks necessary for growth. Contrariwise, hypophysectomy in the rat prevents compensatory hypertrophy, independent of changes in body weight, suggesting that growth hormone is essential for hypertrophy to take place.

The patient we studied was well nourished, gave a history of adequate dietary intake and presented no clinical evidence of endocrine dysfunction. Although the mechanism responsible for the diffuse enlargement of his left kidney is not clear, in all likelihood, it occurred early in life. Could this represent compensatory hypertrophy secondary to intermittent low grade obstruction of the right urinary tract in childhood? Could this be congenital hypertrophy?

B. LOCALIZED (POLAR) ENLARGEMENT OF ONE KIDNEY

There were 2 patients with a pronounced globular appearance of the upper pole of one kidney. In one instance, the right kidney was implicated and in the other, the left. In each case, the well-defined, localized, spherical enlargement of the upper pole suggested the presence of a mass lesion. In this regard, Frimann-Dahl[3] published an example of an enlarged left upper pole, suggesting that this normal variant may occur in the presence of an unusually small spleen. In the authors' case involving the left kidney, the spleen appeared to be normal in size and position.

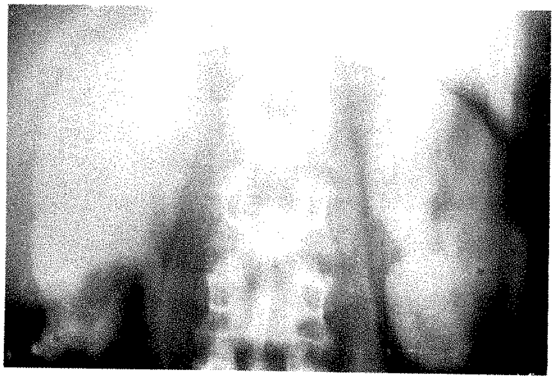

Fig. 13. Case II. Nephrotomogram illustrating enlargement of the upper pole of the right kidney. The patient also had a normal aortogram and normal selective right renal arteriograms. (There were two right renal arteries each of which was selectively catheterized.)

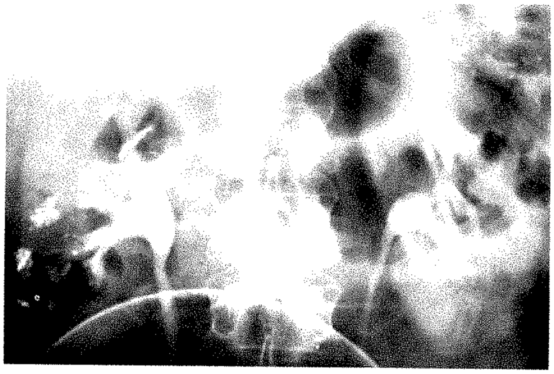

Fig. 14. Case II. Intravenous pyelogram demonstrating a normal pelvicalyceal system.

Case II. R.J., a 54 year old electrician, was admitted to the hospital with acute hepatitis. The patient had no complaints referable to the urinary tract and urinalysis was normal. A plain roentgenogram of the abdomen revealed enlargement of the upper pole of the right kidney. The right kidney measured 16.5 cm. and the left 14.0 cm. in length. Nephrotomography demonstrated a smooth, bulbous upper pole on the right with a normal collecting system excepting for slight elongation of the infundibulum to the upper pole (Fig. 13). Renal arteriography showed two renal arteries on the right but no abnormality of the renal vasculature. The hilus was eccentrically located close to the lower pole, emphasizing the localized upper pole hypertrophy (Fig. 14).

Case III. S.G., a 64 year old white male, fell at home 1 month prior to admission, injuring his left side. One week thereafter, he experienced a single episode of painless hematuria. Physical examination and urinalysis at the time of admission were within normal limits. Retro-

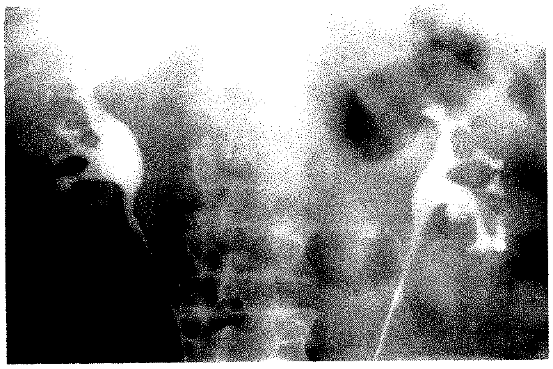

Fig. 15. Case III. Retrograde pyelogram with no gross calyceal distortion (? minimal pyelonephritis). The upper pole of the left kidney was ill-defined and, therefore, further studies were suggested.

grade pyelography demonstrated a normal collecting system bilaterally but the outline of the upper pole of the left kidney was not well visualized. Therefore, nephrotomography and selective left renal arteriography were performed which revealed globular enlargement of the upper pole with normal vasculature (Fig. 15, 16 and 17).

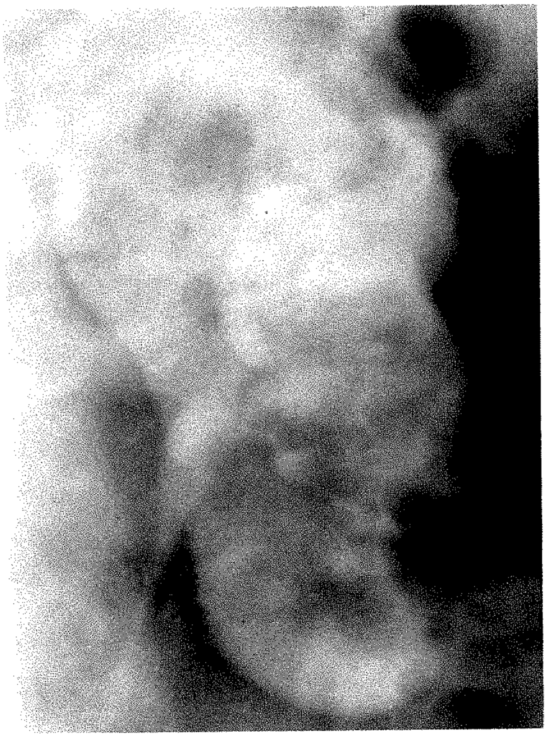

Fig. 16. Case III. Nephrotomographic intravenous pyelogram showing large globular upper pole.

Both patients with globular unipolar renal enlargement had normal intravenous and retrograde pyelograms, nephrotomograms and selective renal arteriograms. In addition, one of the patients (Case III) underwent a surgical exploration and biopsy of the left kidney. At operation, there was no gross evidence of tumor. A biopsy taken from the junction of the lower pole and the enlarged upper pole revealed only mild focal pyelonephritis.

To date, localized globular enlargement of the kidney has not been encountered in the lower pole. If further experience demonstrates that it can also occur in the lower pole, it will have to be differentiated from the artifactual lower pole prominence produced by rotation of the kidney on its transverse axis.[4] This should not pose a

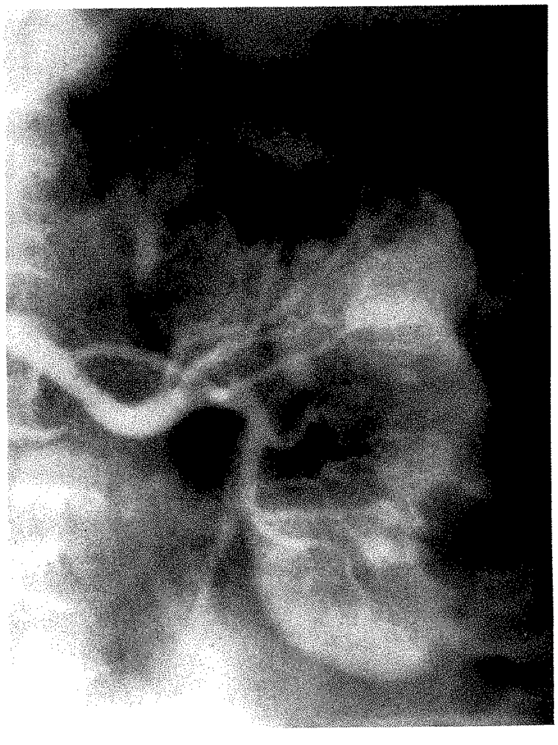

Fig. 17. Case III. Selective left renal arteriogram with normal intrarenal vasculature. Patient had a negative surgical exploration. Biopsy taken from the junction of the enlarged upper pole and the lower half of the kidney showed mild focal pyelonephritis.

significant problem because the abnormal anterior tilt of the lower pole produces the characteristic "bent stem" distortion of the calyceal system and is also readily recognizable on a lateral shoot-through roentgenogram.

The mechanism responsible for localized polar enlargement of one kidney in the presence of a contralateral normal kidney is difficult to understand in the light of present knowledge concerning renal hypertrophy.

III. THE INFLUENCE OF THE LIVER AND SPLEEN UPON RENAL SHAPE

Examination of the viscera *in situ* at necropsy clearly demonstrates that the inferior surface of the liver makes an impression upon the anterior aspect of the right kidney to a varying degree. Occasionally, the inferior surface of the liver is actually hollowed out to form a fossa for the right kidney in the asthenic individual (Fig. 18). The spleen makes a similar indentation on the superior and lateral aspect of the anterior surface of the left kidney. In most patients, the relationship of the liver and spleen to the kidneys does not result in an alteration of the well known normal renal contours. Occasionally, however, variations of the commonly recognized renal shape occur as a result of molding by the liver and spleen. In the latter regard, Frimann-Dahl[3] reported a localized bulge along the lateral margin of the left kidney. This bump, which occurs to a variable degree in approximately 10 per cent of patients, is more commonly localized to the mid segment of the lateral margin of the kidney (Fig. 19). When pronounced, it tends to give the kidney a triangular shape. Harrow and Sloane[6] have picturesquely termed the bump "the dromedary kidney." Frimann-Dahl suggested that the bulge was due to the spleen resting, as it were, upon the superior and lateral margins of the kidney just above the bump. On the other hand, Harrow and Sloane indicate that the bump has no relationship with the spleen. In the authors' experience, the bulge is

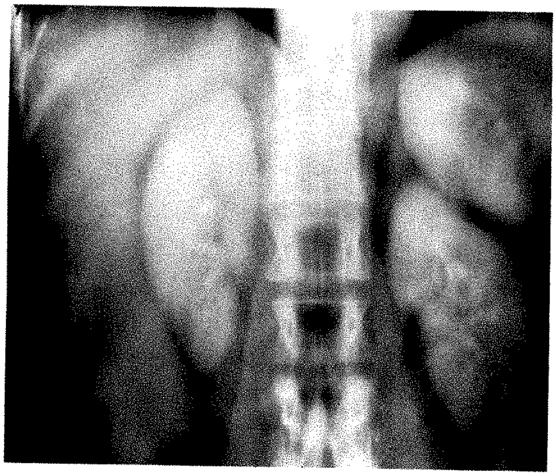

FIG. 18. Nephrotomographic phase of intravenous pyelogram showing the influence of the liver upon the shape of the right kidney in a 55 year old asthenic female.

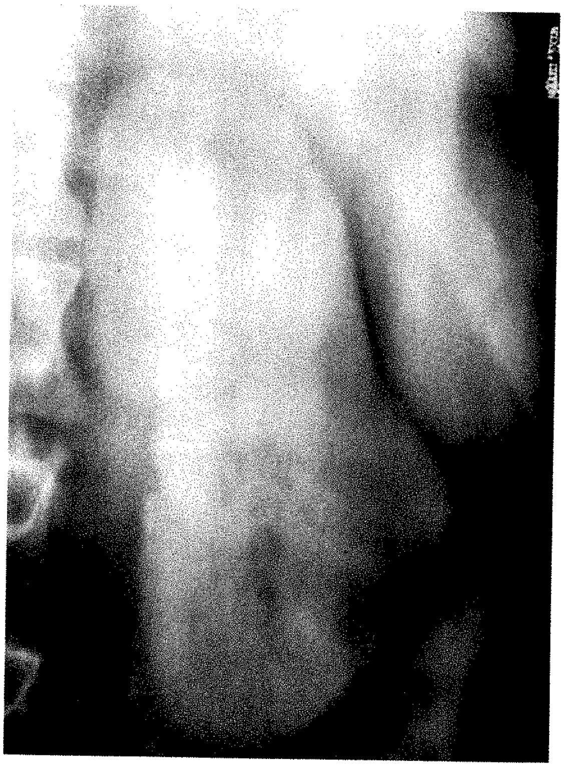

FIG. 19. Nephrotomographic phase of intravenous pyelogram demonstrating the influence of the spleen upon the shape of the left kidney, *i.e.*, "dromedary kidney."

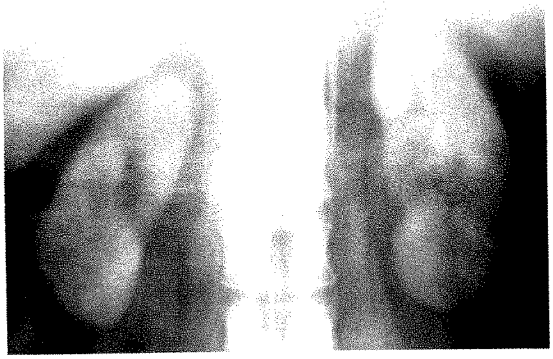

Fig. 20. Nephrotomogram showing a double renal hilus on the left.

usually related to, and produced by, the spleen.

IV. THE DOUBLE RENAL HILUS

The renal sinus is a fat-filled recess in the anterior half of the medial aspect of the kidney containing the renal pelvis, the branching renal arteries, veins and lymphatics. It varies considerably in size. A tongue of renal tissue usually lies behind the renal sinus connecting the posterior halves of the upper and lower poles. The renal sinus is best visualized by nephrotomography with the patient in the prone position. Under these conditions, the kidney tends to lie in a true coronal plane. The renal sinus is not well visualized in the supine position in those patients with an anterior renal hilus due to rotation of the kidney on its longitudinal axis.

Although the hilus is usually central in location, it may be eccentric, lying closer to either the upper or lower pole. When there is duplication of the collecting system, two hili can often be visualized on the nephrotomogram. In our experience to date, there does not seem to be any relationship between a double hilus and multiple renal arteries.

Demonstration of a double hilus is helpful in the diagnosis of renal duplication when one of the collecting systems fails to visualize (Fig. 20, 21, 22, 23 and 24). Windholz,[16] in his classic paper on the central fatty tissue of the kidney, suggested that the presence of intrarenal fat outside the opacified collecting system may be helpful in predicting the presence of an additional nonfunctioning duplication. Visualization of a double renal hilus by nephrotomogra-

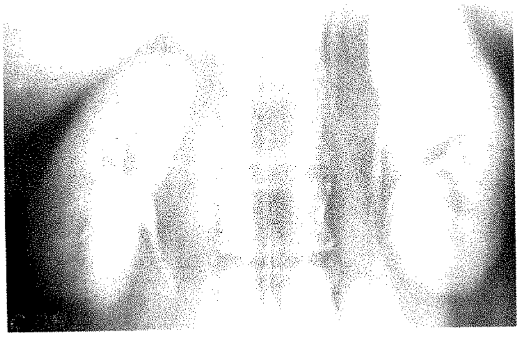

Fig. 21. Pyelographic phase illustrating the duplication of the left kidney pelvis and calyces in the same patient as in Figure 20.

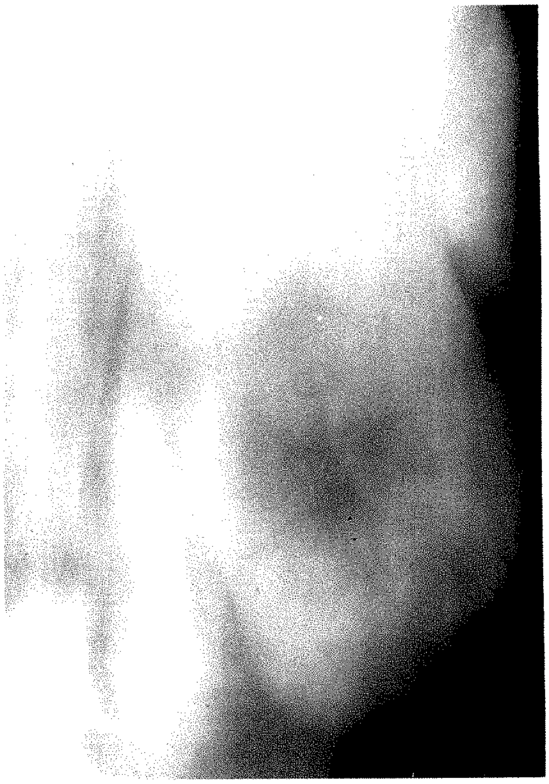

Fig. 22. Nephrotomogram demonstrating a double left renal hilus. Note the dual collections of peripelvic fat and the double indentation along the medial margin of the kidney.

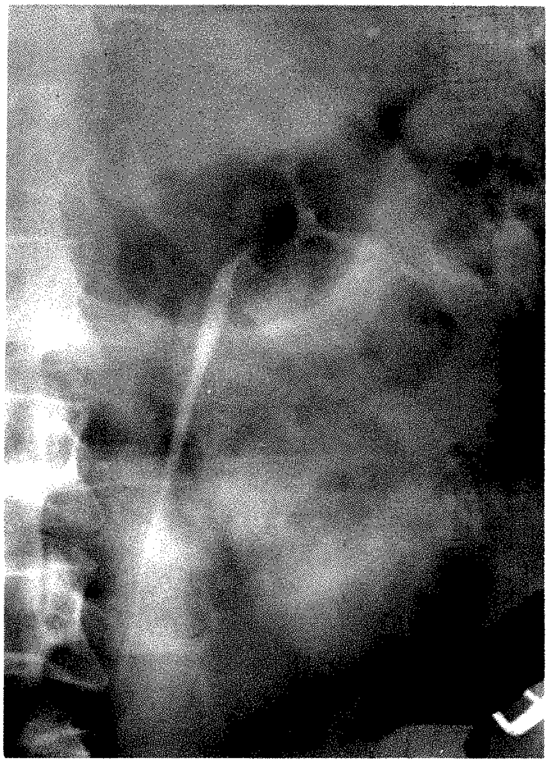

Fig. 23. Retrograde pyelogram showing filling of the upper calyceal system.

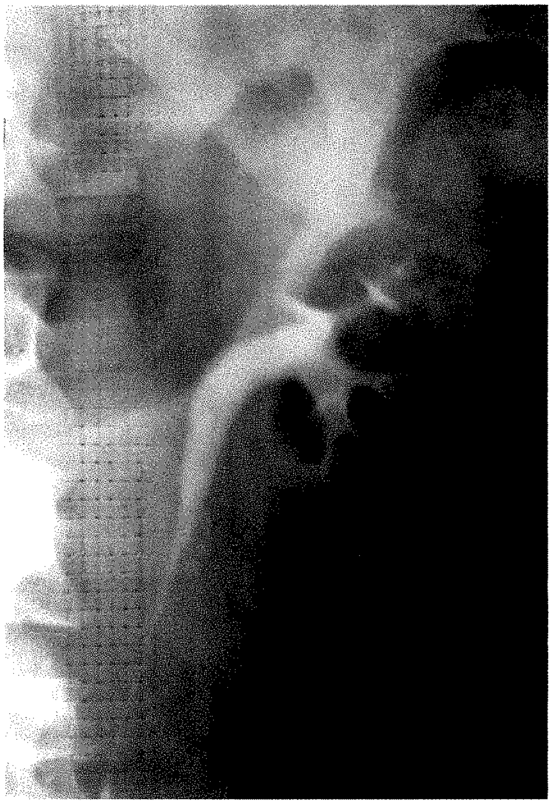

Fig. 24. Retrograde pyelogram showing filling of the lower calyceal system.

phy serves a similar purpose, provided that there is some residual function in the duplicated segment.

SUMMARY

1. The authors have described several normal variants which may be confused with renal mass lesions, *i.e.*, suprahilar bulge, and diffuse and localized enlargement of the kidney.

2. The relationship of the liver and spleen to renal shape is discussed.

3. The double hilus sign and its significance are also discussed.

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INFUSION PYELOGRAPHY*

By JOHN H. HARRIS, M.D., *and* JOHN H. HARRIS, Jr., M.D., D.Sc. (Med.)†
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INFUSION pyelography is the term applied to the technique of opacifying the urinary tract by means of large amounts of contrast medium introduced by rapid continuous intravenous drip.[9] This procedure has proven to be an extremely dependable, safe method of studying the urinary tract, combining the advantages of the routine intravenous and retrograde studies without the inconveniences and inherent potential hazard of the latter.

TECHNIQUE

One hundred and forty cubic centimeters of 50 per cent hypaque sodium (diatrizoate sodium) are mixed with an equal amount of physiologic salt solution and allowed to run freely through an 18 gauge hypodermic needle placed in an antecubital vein. About 6 minutes is required for 225 cc. of the mixture to enter the vein, at which time the rate of infusion is slowed in order to maintain an open needle for the duration of the

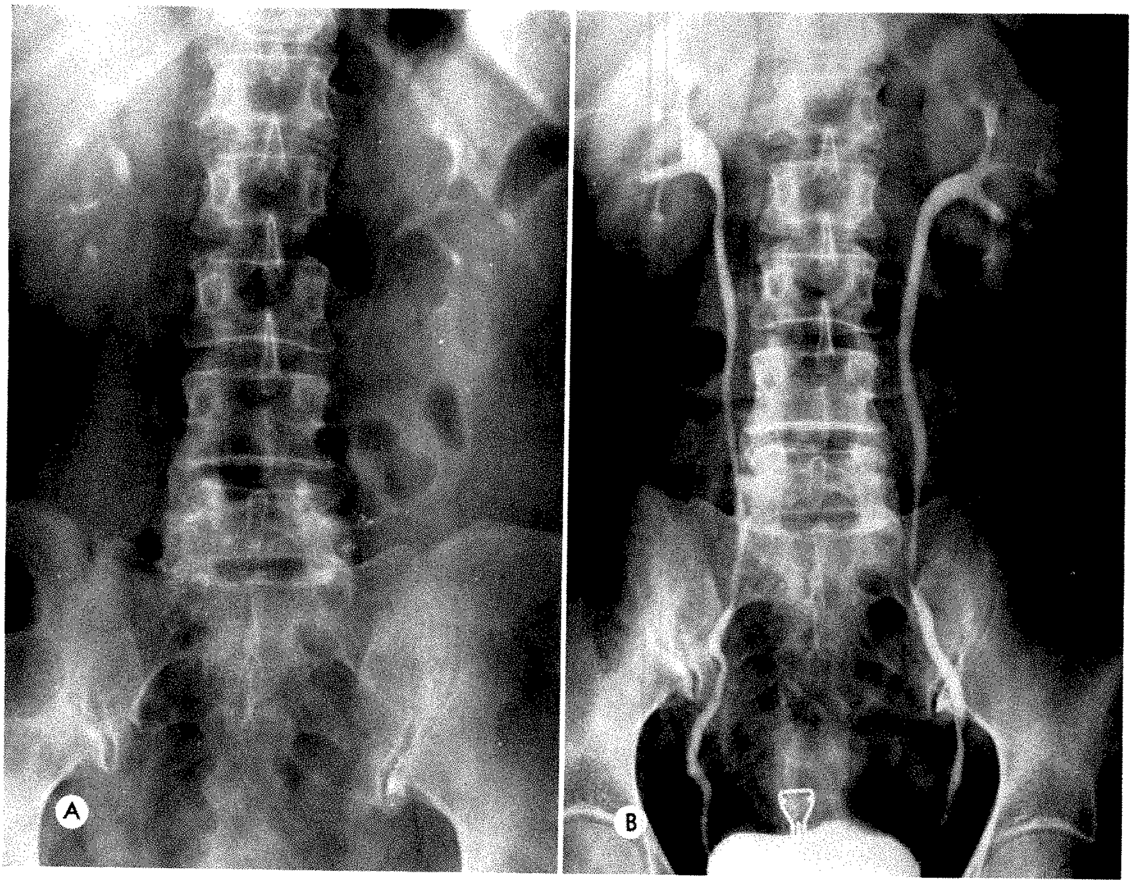

Fig. 1. A 60 year old female with Stage II carcinoma of the cervix. (*A*) Routine intravenous pyelogram. (*B*) Infusion pyelogram demonstrates renal morphology, ureteral course, and bladder configuration. Retrograde pyelography was not necessary.

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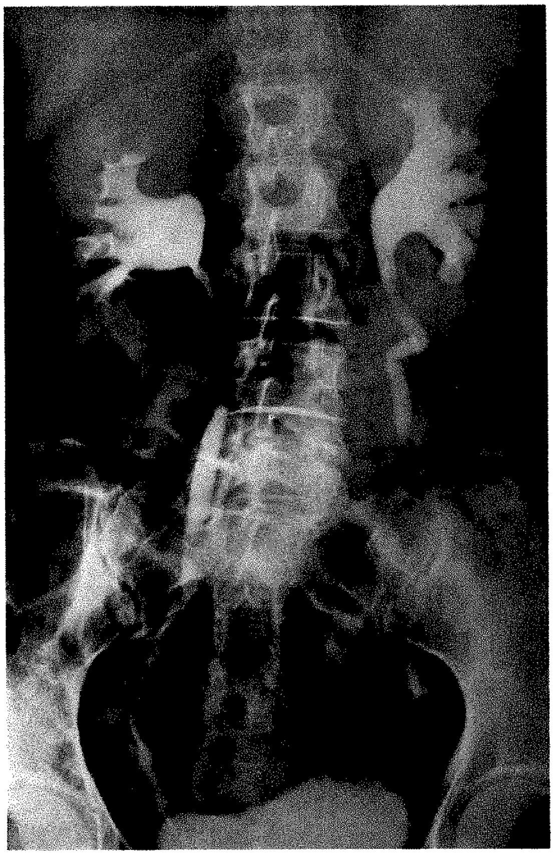

FIG. 2. A 55 year old white female with simultaneous Stage III carcinoma of the cervix (extension to right pelvic wall) and carcinoma of sigmoid colon. Infusion pyelogram demonstrates normal right renal function, displacement of the right ureter, minor left hydronephrosis, and displacement of the left ureter by the colon tumor. Retrograde studies were not necessary.

study. The first roentgenogram is made 20 minutes following commencement of the infusion. Subsequent studies may include, in addition to routine frontal projections, appropriate oblique roentgenograms and laminagrams.

DISCUSSION

The visualization of the renal collecting systems by routine intravenous administration of the usual amounts of contrast medium is variable with the most stringent of techniques. When the technical quality of the examination decreases, the diagnostic quality of the examination is further impaired. Also, in certain pathologic conditions such as renal disease, hypertension, or obstructive uropathy, the detailed morphology of the collecting system is frequently not well demonstrated by routine urography.

During routine intravenous urography, the ureters are "normally" incompletely opacified. In fact, complete opacification of an entire ureter is commonly accepted as being highly indicative of a pathologic state of the ureter or bladder. However, there are many occasions when it is desirable to demonstrate the course of the ureters completely, *i.e.*, ureteral displacement by enlarged retroperitoneal lymph nodes, ureteral neoplasm, the preoperative and post-

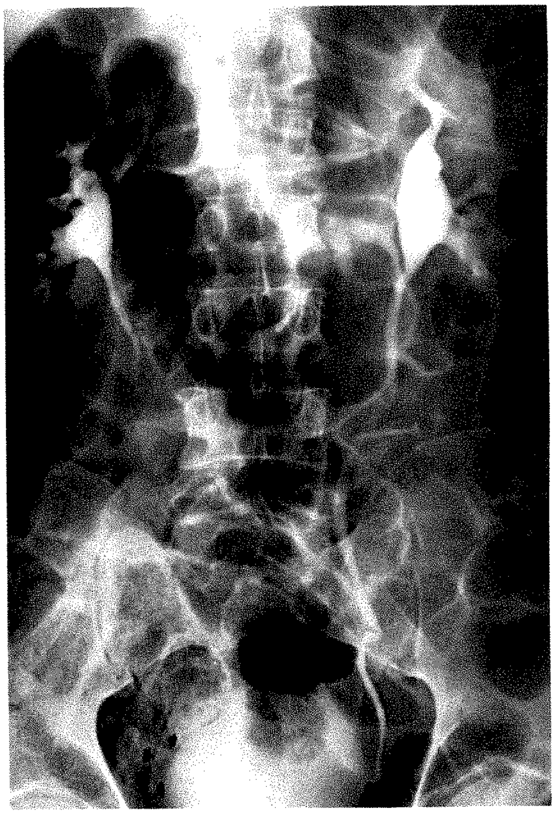

FIG. 3. This roentgenogram demonstrates the value of infusion pyelography in the unprepared patient or in the presence of intestinal distention. The differential diagnosis in this 50 year old male included right ureteral calculus or acute appendicitis. The urinary tract is normal. Arrows define the course of the distal right ureter.

operative evaluation of pelvic masses, the status of the ureters in association with adnexal involvement by advanced cervical carcinoma, and neoplasms or diverticulosis of the sigmoid colon.

When the urinary tract is not optimally opacified by routine intravenous methods, it has been customary to resort to retrograde pyelography or one of the renal arterial injection techniques. Each of these examinations can provide better opacification of the collecting system and, with specific indications, each serves a valuable purpose. However, both procedures have definite limitations, require hospital admission, and are attendant with occasional complications.[1,2,5,6,7,10] Patient motion, technically inferior pyelograms, the difficulty of obtaining oblique roentgenograms and/or laminagrams in association with the retrograde examination, incomplete filling, overdistention of the collecting systems, and the introduction of air are but a few of the many obstacles to obtaining retrograde studies of diagnostic caliber. In addition, retrograde studies require urinary tract instrumentation.[8]

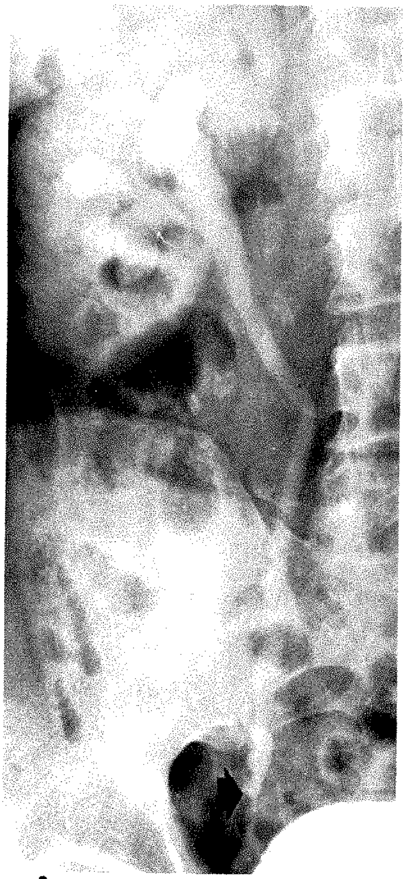

Fig. 4. The incompletely obstructing distal right ureteral calculus (arrow) was located by infusion pyelography 40 minutes after the initiation of the study.

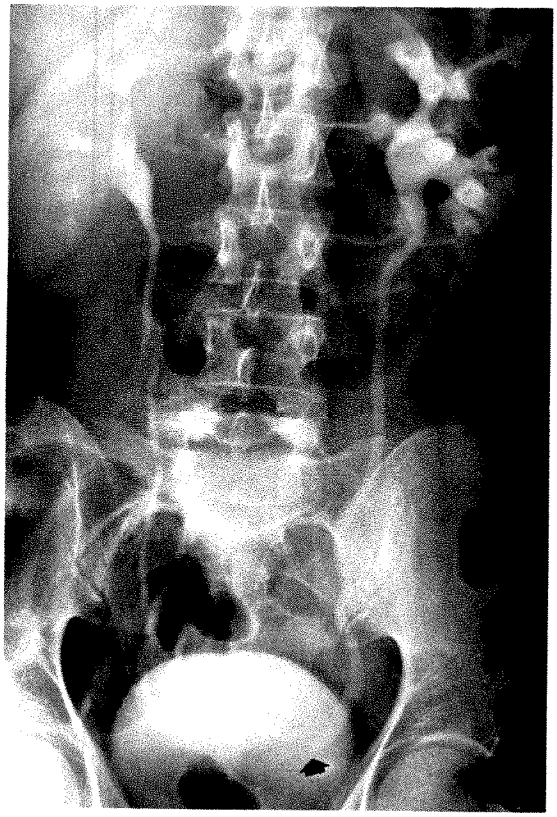

Fig. 5. The obstructing calculus at the left ureteral-vesicle junction (arrow) was demonstrated 20 minutes following infusion of the contrast medium.

Renal arteriography or aortography also usually requires hospital admission. While greater numbers of radiology departments are being equipped with pressure injection and rapid film changing systems, arterial injection studies are still not commonly available outside of metropolitan institutions. Furthermore, the rapidity of the passage of the contrast medium through the kidneys makes it difficult to do more than

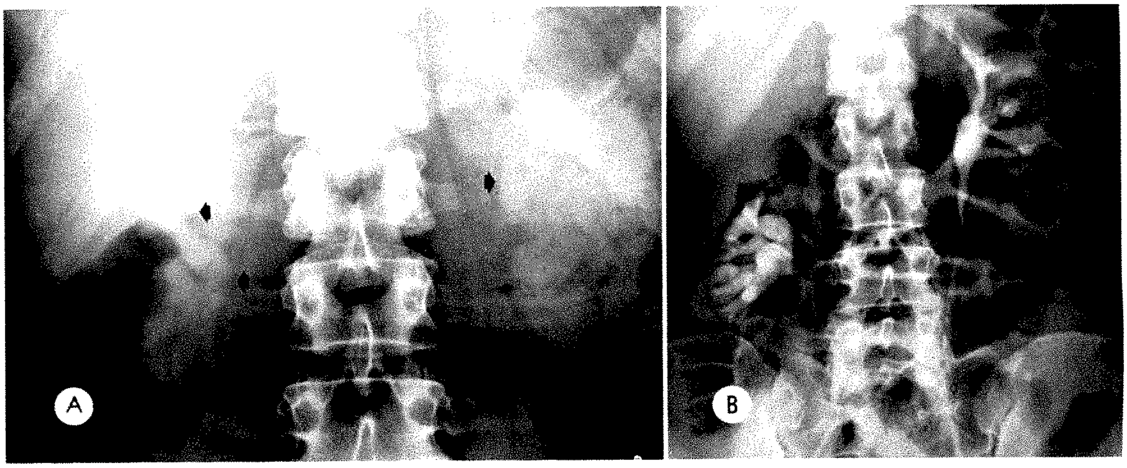

Fig. 6. These studies illustrate the value of infusion pyelography in hypertensive patients. (*A*) Routine pyelogram demonstrates maximum visualization of the kidneys. The faintly opacified collecting systems are indicated by arrows. (*B*) Infusion pyelogram obtained 24 hours later.

one type of roentgenographic examination per injection.

In order to obtain qualitatively better studies of the urinary tract, without the problems inherent in retrograde or arterial studies, we performed infusion pyelographies on 50 patients to date. In all but 1 patient in whom there was a congenitally absent left kidney, the infusion pyelogram was diagnostically superior to the routine intravenous study, and in none of these patients was retrograde pyelography necessary. The infusion studies routinely delineated the entire urinary system, usually within 20–30 minutes following completion of the infusion. Because of complete opacification of each ureter, retrograde studies were not necessary in the evaluation of pelvic masses. The pelvicalyceal systems remained opacified long enough to permit obtaining roentgenograms in oblique projection and/or laminagrams when indicated. Maximum visualization of the urinary system, without artifacts, was routinely achieved whether the patient had been dehydrated and the colon evacuated

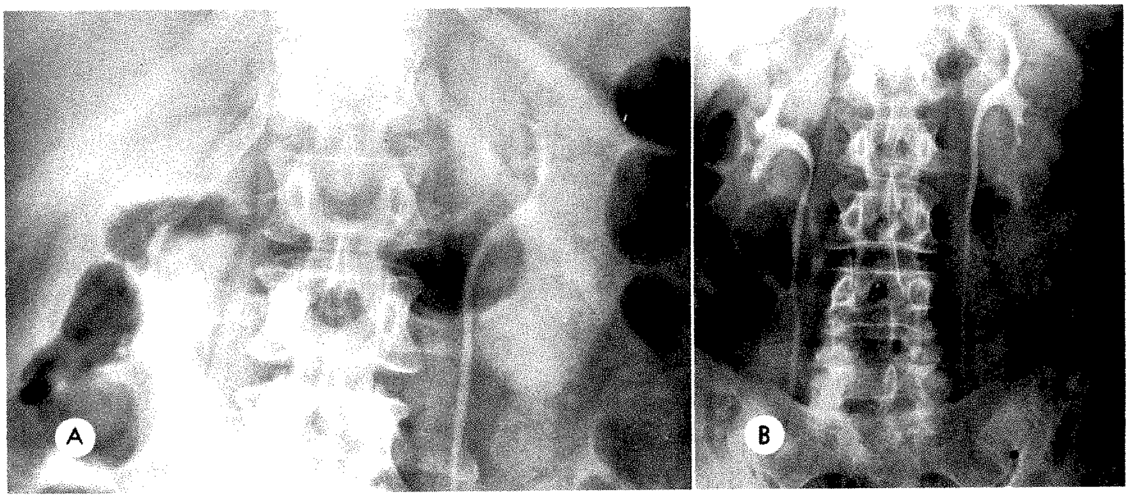

Fig. 7. (*A* and *B*) These figures contrast the degree of opacification of the urinary tract that was obtained by (*A*) routine intravenous pyelography and (*B*) infusion pyelography. The studies were done 6 hours apart on the same patient.

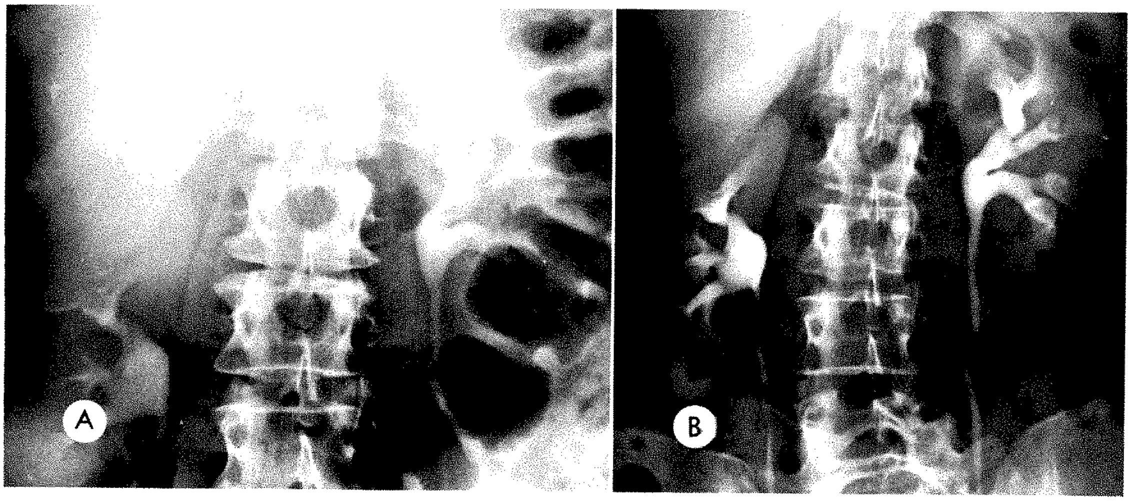

Fig. 8. (*A*) Routine intravenous pyelogram and (*B*) infusion pyelogram obtained 5 hours apart on the same patient.

or not. In this small group of patients, there have been no allergic reactions following infusion of 140 cc. of contrast medium. Infusion pyelography has been performed immediately following routine intravenous urography, later the same day, and 24–48 hours following the intravenous study. It has been interesting to observe that in those patients experiencing mild, transient allergic responses to routine intravenous administration of 25 cc. of 50 per cent hypaque, no reaction was precipitated during, or following, the rapid infusion of 140 cc. of 50 per cent hypaque diluted with an equal volume of physiologic salt solution. Infusion pyelographies are performed on an outpatient basis.

Figures 1 through 10 demonstrate the diagnostic advantages of infusion pyelograms in comparison to routine intravenous urograms.

SUMMARY

Pyelographic studies employing rapid infusion of 140 cc. of 50 per cent hypaque

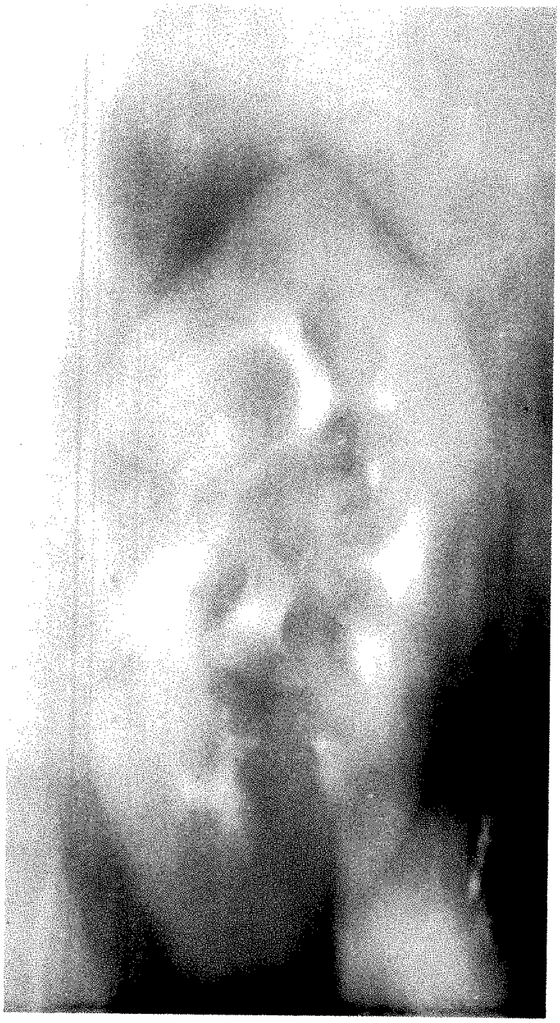

Fig. 9. Laminagram with infusion pyelography demonstrates the classic findings of multiple cysts of the kidney.

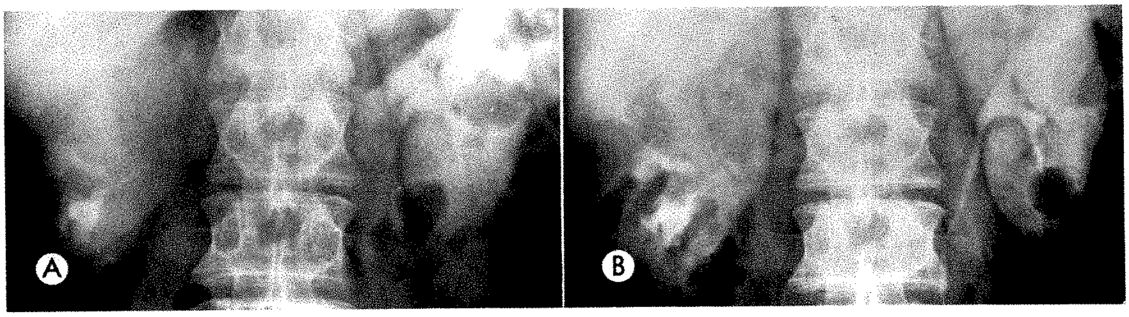

Fig. 10. Hypernephroma of right kidney. *A* was made 2 hours following routine intravenous pyelography. The distorted right collecting system was incompletely seen; the left was only fairly demonstrated. *B* is the pyelogram made 15 minutes following completion of the infusion.

diluted in equal volumes of physiologic salt solution provide markedly improved demonstration of the renal collecting systems when routine intravenous urograms are not diagnostic. Uniformly optimum opacification of the ureters has greatly reduced the necessity of retrograde pyelography in evaluation of pelvic tumors. Because of prompt and dense delineation of the urinary system, infusion pyelography is of distinct advantage in studying the patient with ureteral colic, or the patient in whom the gut contains large amounts of gas or intestinal debris. The prolonged infusion of the contrast medium permits a leisurely study of the kidneys by oblique roentgenograms and/or laminagrams when indicated. Infusion nephrotomography aids in differentiating renal cysts and neoplasms[3,4] without the inconvenience and inherent dangers of aortography or renal arteriography.

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THE VALUE OF THE UREA WASHOUT TEST IN THE ASSESSMENT OF CURABLE RENOVASCULAR HYPERTENSION*

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IN 1962 a preliminary report was published by one of us (K.A.) describing a new roentgenographic technique for assessing renovascular hypertension, namely, the urea washout intravenous pyelography.[1] Since that time, much more experience has been gained with this procedure. It is the purpose of the authors to report results and to critically evaluate the usefulness of the urea washout test in the assessment of curable renovascular hypertension.

MATERIAL

A total of 463 hypertensive patients from the University of Minnesota Hospitals, Minneapolis Veterans Administration Hospital and Ancker Hospital, St. Paul, Minnesota, were examined from May 21, 1962 through March 21, 1964. Patients were unselected as to type of hypertension. The age range was from 12 to 85 years. A total of 488 washout tests were performed on this group, including those done postoperatively and several repeat studies done because of equivocal results. Excluded were patients concurrently under treatment for congestive heart failure, patients with suspected iodide sensitivity, and patients with severely impaired renal function to the degree that opacification of the renal collecting systems could not be expected. Radioactive renograms were obtained in 145 patients, renal arteriograms in 108 patients, and split renal function studies and the Rapoport test[18] were performed on 12 patients. A total of 16 patients had surgery, 12 with positive washout tests and 4 with normal tests.

TECHNIQUE AND INTERPRETATION

1. The patient is prepared as for a routine excretory urography, including the customary period of dehydration.
2. No compression device is used in order to allow unhindered emptying of the collecting systems.
3. Following the scout roentgenogram, 50 ml. of a more highly concentrated diatrizoate compound (75 per cent hypaque or 76 per cent renografin) is manually injected intravenously as rapidly as possible through a No. 19 gauge needle. A large antecubital vein is usually employed.
4. The needle is left in place and secured and to it is connected a very slowly dripping infusion set containing 500 ml. of normal saline and 40 gm. of urea.* The slow rate of infusion at this time is intended to merely keep the needle open.
5. Pyelograms are made at $\frac{1}{2}$, 1, 3, 5 and 8 minutes following the injection of contrast medium. The 3 minute film is immediately developed and viewed to establish the presence of adequate kidney function.
6. Following exposure of the 8 minute pyelogram at which time the renal collecting systems are usually optimally opacified, the saline-urea solution is rapidly infused in 10 to 15 minutes.
7. From the moment that the rapid infusion is started, films are exposed at 3 minute intervals to demonstrate the unilateral or bilateral dilution of the renal

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* Mixture is prepared by adding 50–100 ml. of saline from the 500 ml. normal saline bottle to the bottle containing 40 gm. of urea (Ureaphil, Abbott Laboratories). Allow urea to dissolve and reinject the solution into the 500 ml. normal saline bottle.

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This study was supported in part by USPHS Grants HTS-5222 (C 4) and HE-06314-04.

collecting systems which occurs. In either case this is usually conclusive by the 18 minute postinfusion pyelogram at which time the procedure is routinely terminated. Essentially equal bilateral dilution occurs in a normal washout test.

If there is extremely poor concentration bilaterally, the saline-urea infusion is not started, thus excluding patients with poor kidney function. This also applies in the case of a unilaterally nonfunctioning kidney. The exception to this is the case showing markedly delayed appearance of contrast agent on one side, which may indicate a very severe renal artery stenosis. If given time, the contrast density will eventually increase in this situation and hyperconcentration will result. If such is suspected while viewing the pre-infusion pyelograms, a reinjection of contrast medium and an additional pyelogram at 12 or 15 minutes may be helpful prior to starting the saline-urea infusion.

By using a larger amount of iodine than is used in standard urographic examinations, the nephrographic effect is enhanced and excellent opacification of the collecting systems results, giving good anatomic detail without compression. The value of early pyelograms following the rapid injection of contrast medium in detecting renal artery stenosis has been well shown by other investigators.[12] The most important physiologic alteration of curable renovascular hypertension, namely, hyperconcentration due to excessive tubular water reabsorption,[3,23] is dramatically demonstrated by suddenly induced diuresis following opacification of the renal collecting systems.

POSITIVE WASHOUT

A positive washout is characterized by a striking difference in opacification of the renal collecting systems following the infusion of saline and urea. While the collecting system of the normal kidney becomes rapidly and progressively less dense during the saline-urea infusion, that of the ischemic kidney remains denser and "washes out" much more slowly. It is to be emphasized that this discrepancy should be apparent on all the postinfusion pyelograms. This characteristic demonstration of the altered physiology is commonly associated with some of the classic roentgenographic findings of renovascular hypertension, namely, decreased kidney size, delayed nephrogram, increased or decreased nephrographic effect, delayed excretion of contrast medium and a smaller collecting system.[5,9,10,15,19] Features of a positive washout test are shown in Figure 1, *A–E*.

PSEUDOPOSITIVE WASHOUT

Occasionally, a washout test will appear questionably positive or equivocal due to the following reasons:

1. A unilateral larger renal collecting system, usually an extrarenal pelvis, which will show a delayed clearance of contrast medium during the saline-urea infusion due to its larger capacity (Fig. 2, *A* and *B*).
2. Apparent decreased density of one renal collecting system because of overlying intestinal contents. Comparison between the two sides becomes particularly difficult if gas shadows move over one kidney during the postinfusion phase of the examination (Fig. 3, *A* and *B*).
3. Peristaltic contractions of one renal pelvis at the time of exposures of the postinfusion films resulting in decreased density of the pelvis. This is normally seen only on 1 or 2 films (Fig. 4, *A* and *B*).
4. Rotation of one kidney resulting in its increased density on all pre- and postinfusion pyelograms due to the roentgen rays passing through a longer diameter of the opacified pelvis (Fig. 5, *A* and *B*).
5. Ureteropelvic junction or ureteral obstruction which results in delayed emptying on one side (Fig. 6, *A* and *B*).
6. Unilateral renal parenchymal disease, as pyelonephritis, causing a decreased density of one collecting system on the pre-infusion pyelograms and, as a result, washing out relatively faster than does the opposite normal kidney. The washout of the normal kidney will not be delayed, how-

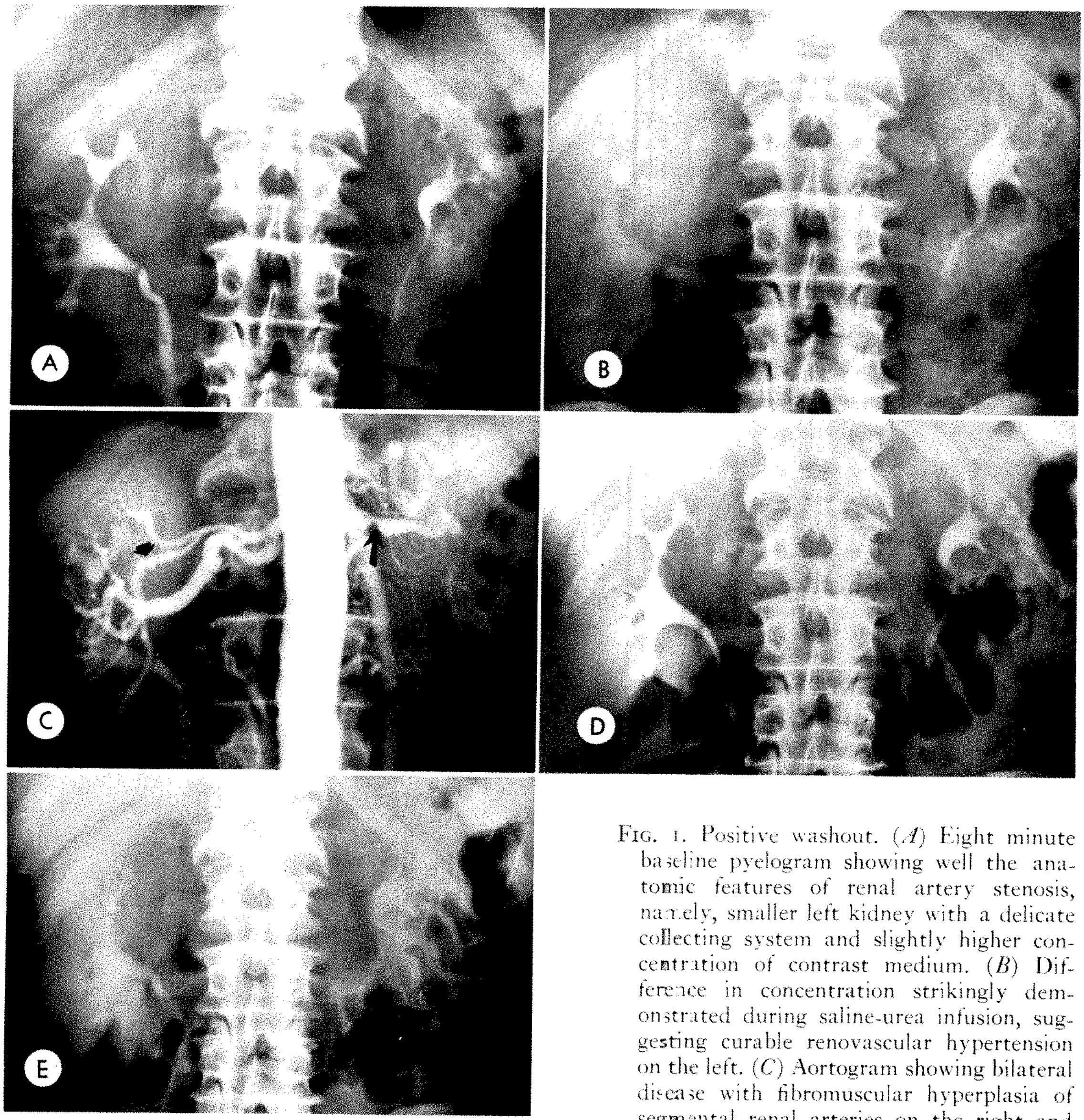

Fig. 1. Positive washout. (*A*) Eight minute baseline pyelogram showing well the anatomic features of renal artery stenosis, namely, smaller left kidney with a delicate collecting system and slightly higher concentration of contrast medium. (*B*) Difference in concentration strikingly demonstrated during saline-urea infusion, suggesting curable renovascular hypertension on the left. (*C*) Aortogram showing bilateral disease with fibromuscular hyperplasia of segmental renal arteries on the right and severe stenosis of the left renal artery by atherosclerotic plaque. Note delayed blood flow through stenosed left renal artery. Positive urea washout test on left suggested that left renal artery stenosis was responsible for patient's hypertension and bilateral surgery, therefore, was not indicated. (*D*) Following surgery, patient's blood pressure returned to normal. Baseline 8 minute pyelogram of postoperative washout test. (*E*) Bilaterally equal dilution on postinfusion pyelogram (22 minutes from injection of contrast medium) of postoperative washout test.

ever, as would be the case in a true positive test. There are usually also anatomic changes suggesting pyelonephritis.

If one or more of these features is present, a pseudopositive study should be suspected and the test must be interpreted with caution. In certain of these cases, it will be sufficient to merely repeat the study on another day if the washout is equivocal. Renal arteriography should be performed in all doubtful cases. In our experience, however, a truely positive washout with significant unilateral renal artery stenosis on arteriography was always a very striking

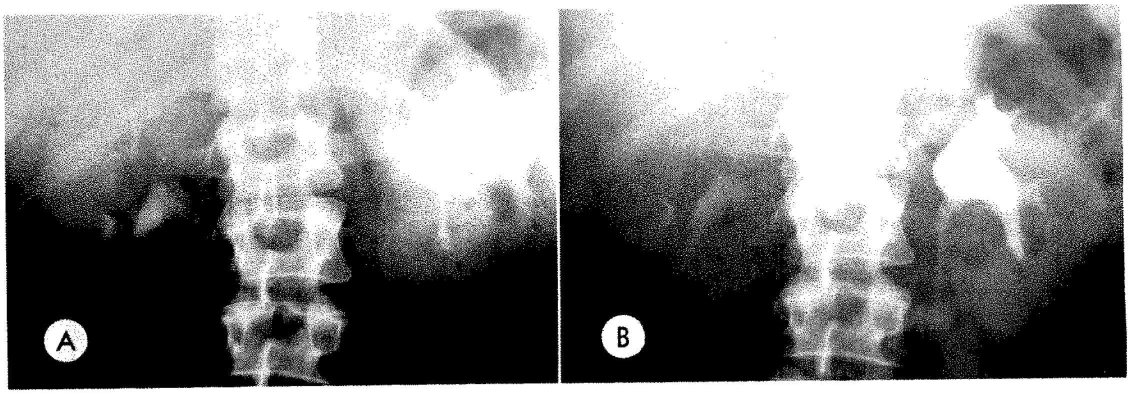

Fig. 2. Pseudopositive washout. (*A*) Three minute pyelogram showing delayed opacification of left collecting system which contains more urine due to its larger size. (*B*) Twelve minute pyelogram after start of saline-urea infusion demonstrating almost complete clearing on the right, but delayed clearing of left collecting system which has accumulated a larger amount of contrast medium due to its greater capacity.

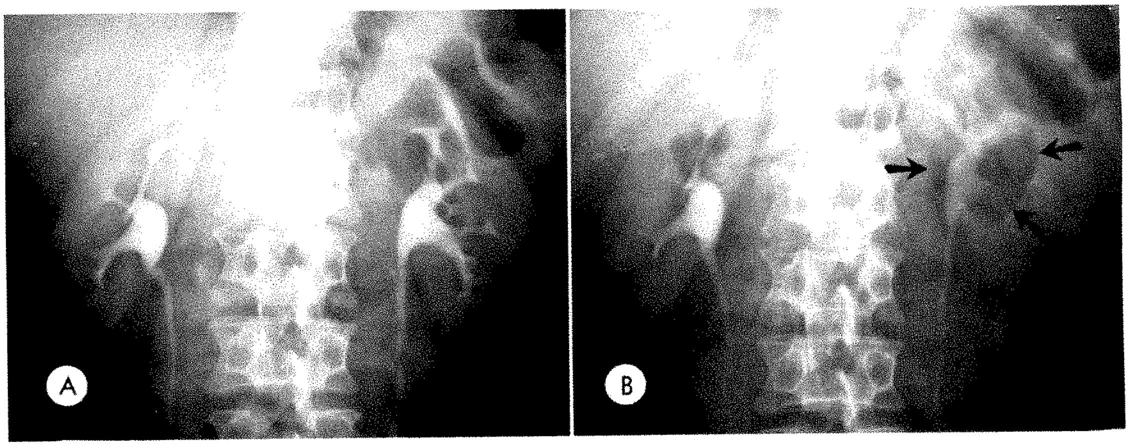

Fig. 3. Pseudopositive washout. (*A*) Eight minute baseline pyelogram showing equal opacification of collecting systems. (*B*) During saline-urea infusion, gas shadows have moved over left collecting system simulating positive washout on right.

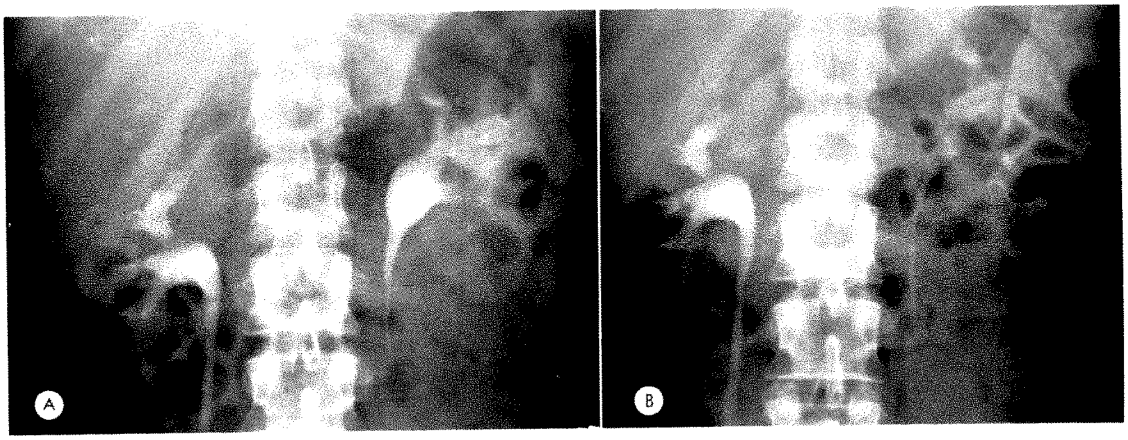

Fig. 4. Pseudopositive washout. (*A*) Eight minute baseline pyelogram showing equal opacification of collecting systems. (*B*) Peristaltic contraction of left collecting system simulating positive washout on left. Reappearance of a larger and denser collecting system on subsequent pyelograms should suggest a pseudopositive washout due to peristaltic contraction.

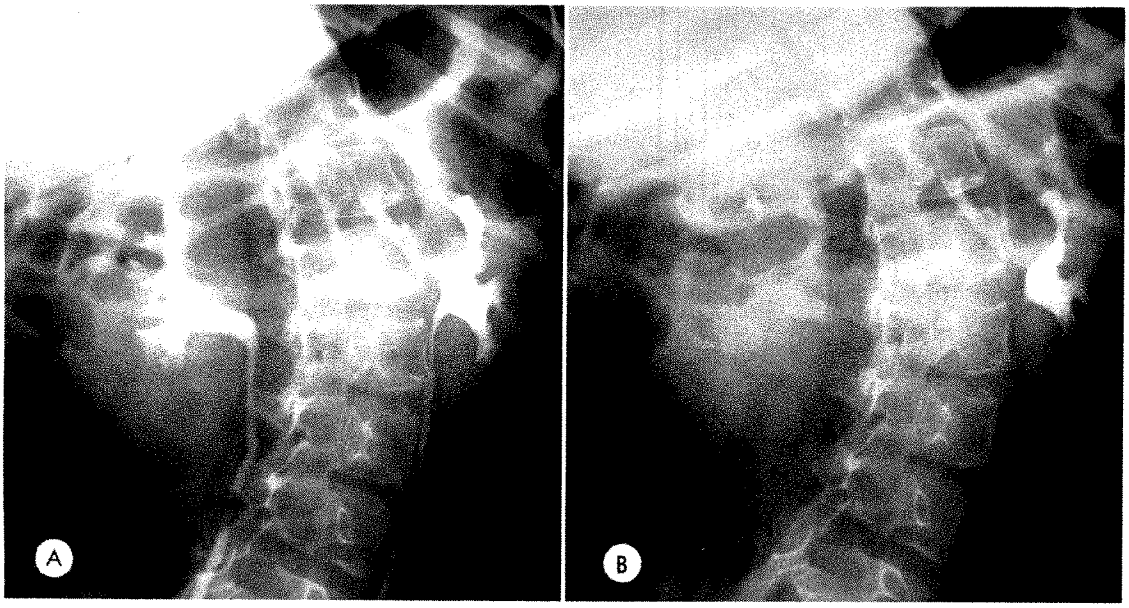

Fig. 5. Pseudopositive washout. (*A*) Marked rotation of left kidney due to scoliosis simulating hyperconcentration on 8 minute baseline pyelogram. (*B*) Positive washout simulated by rotation of left kidney.

finding and left no question in interpretation.

COMPLICATIONS

There were no serious complications encountered, although headaches did occur in approximately 20 per cent of the cases during or immediately after the saline-urea infusion. This was successfully controlled by the administration of aspirin and several glasses of water following termination of the procedure. Rarely, transient tremulousness or chills occurred during the saline-urea infusion. The incidence of nausea, vomiting, or other discomfort following the rapid injection of contrast medium was extremely low.

RESULTS

The urea washout tests of 463 hypertensive patients were reviewed (Table 1). The findings were correlated with radio-

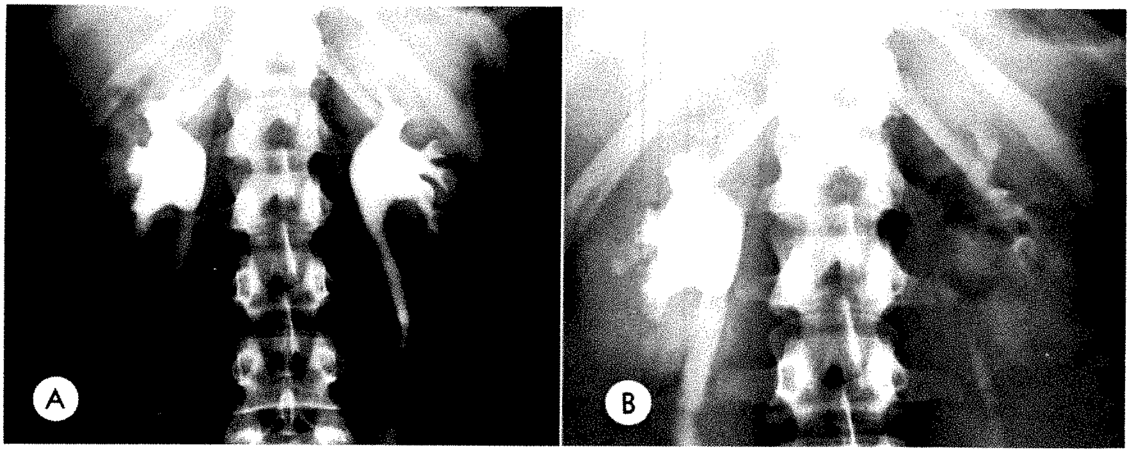

Fig. 6. Pseudopositive washout. (*A*) Eight minute baseline pyelogram showing equal opacification of collecting systems. (*B*) Following saline-urea infusion, delayed positive washout on right simulated by obstructive uropathy with considerable ureterectasis.

TABLE I

STUDIES ON 463 HYPERTENSIVE PATIENTS

Studies	Total Performed	Studies on 15 Patients with Positive Urea Washout Tests		
		Total	Positive	Negative
Renal Arteriograms	108	15	14	1
Radioactive Renograms	145	10	9	1*
Split Renal Function Tests	12	5	4	1

* Not typical stenosis pattern. Unilateral depressed function suggesting parenchymal disease.

active renograms and renal arteriograms which were obtained on all patients with a high clinical suspicion of renovascular hypertension, regardless of the results of the washout test or radioactive renogram. Fifteen positive tests were found and in each patient, except 1, a severe renal artery stenosis was demonstrated angiographically. The single patient having a positive washout test and no renal artery stenosis represents a false positive result.

Pseudopositive washout tests due to one of the previously described factors were found in 17 patients, 15 of whom had arteriography with no significant renal artery stenoses found. Minor stenoses felt to be of no hemodynamic significance were found in 2 patients. In 13 patients it was necessary to perform arteriography in order to definitely exclude renal artery stenosis because of the equivocal washout findings, although a pseudopositive test was suspected in each case. Repeating the washout studies was sufficient in the remaining patients. The unilateral larger renal collecting system proved to cause the most difficulty in interpretation.

Abnormal radioactive renograms were found in 61 of 145 examined patients. Thirty-one of the total 145 patients had renal arteriograms. In 4 patients with proven renal artery stenoses, normal renograms were obtained. Only 1 of these was of a significant degree, however. This patient also had a normal washout test and no corrective surgery has been performed. Of the 61 abnormal radioactive renograms, 25 showed a rather typical stenosis pattern as indicated by a slight delay in vascular rise, a significant delay in peak activity and delayed excretion. This pattern was present in 9 of the 10 positive washout cases having radioactive renograms (Table II). Of the remaining 16 cases with positive radioactive renograms, but with normal washout test, arteriograms were obtained on 9 patients and renal artery lesions were found in all, 4 believed to be of a significant degree.

Split renal function studies were performed on 5 of the positive washout cases

TABLE II

RESULTS OF 145 RADIOACTIVE RENOGRAMS IN EVALUATION OF HYPERTENSIVE PATIENTS

Radioactive Renograms	No. of Patients	Washout Tests		Renal Arteriograms			
		Positive	Negative	Total	Total Stenoses	Significant Stenoses	Normal
Normal	84	0	84	9	4	1	5
Abnormal	61						
(a) Stenosis pattern	25	9	16	18	18	4	0
(b) Not typical stenosis pattern	36	1	35	4	2	1	2

TABLE III

SURGICAL RESULTS IN 12 POSITIVE UREA WASHOUT PATIENTS

Patient	Urea Washout	Renogram	Split Renal Function Test	Renal Arteriogram	Surgery	Result
W.K.	Positive lt.	Positive lt.	None	Atherosclerotic plaque in lt. main renal artery	Endarterectomy with patch graft	Normotensive
P.V.	Positive lt.	Positive lt.	Positive lt.	Atherosclerotic plaque in lt. main renal artery	Bypass graft	Normotensive
Y.A.	Positive rt.	None	Positive rt.	Severe stenosis in rt. main renal artery	Endarterectomy with patch graft	Normotensive
J.I.	Positive lt.	Abnormal lt; not typical stenosis	Normal	Atherosclerotic plaque in lt. main renal artery	Bypass graft	Normotensive
C.J.	Positive rt.	Positive rt.	None	Fibromuscular hyperplasia in rt. renal artery	Bypass graft	Normotensive
J.C.	Positive rt.	Positive rt.	None	Atherosclerotic plaque in rt. main renal artery	Endarterectomy with patch graft	Normotensive
O.G.	Positive rt.	None	None	Severe stenosis in rt. main renal artery	Bypass graft	Normotensive
J.T.	Positive lt.	Positive lt.	Positive lt.	Atherosclerotic plaque in lt. main renal artery	Difficult endarterectomy; patch graft	Improved but still has mild hypertension
E.B.	Positive lt.	Positive lt.	None	Almost complete occlusion in lt. renal artery	Nephrectomy	Improved but still has mild hypertension
L.R.	Positive lt.	None	None	Atherosclerotic plaque in both lt. segmental renal arteries	Bypass graft	Improved but still has mild hypertension
J.L.	Positive lt.	None	None	Atherosclerotic plaque in lt. main renal artery	Bypass graft	Improved but has persistent mild hypertension; recent surgery with only 2 weeks follow-up
C.L.	Positive rt.	Positive rt.	Positive rt.	Fibromuscular hyperplasia in large rt. artery segmental renal artery	Vein graft	No improvement; developed renal infarct due to occlusion of vein graft

and excellent correlation between the urea washout and the Rapoport test was found in 4 patients. One patient was said to have a normal Rapoport test, although he was cured following surgical correction of his renal artery stenosis. Seven patients with renal artery stenoses and negative washout tests also had normal split renal function tests.

Twelve patients from the positive washout group had corrective surgery consisting of bypass grafts in 6, endarterectomies with patch grafts in 4, vein graft in 1 and nephrectomy in 1 (Table III). Seven patients became normotensive following surgery and have remained so without use of antihypertensive drugs. Postoperative intervals for these patients now range from 4 months to 2 years. Repeat washout tests done postoperatively all became normal. In 3 of the remaining 5 patients, the hypertension improved considerably following surgery, but the blood pressure has not completely returned to normal. A fourth patient, presently considered as improved rather than cured, has not as yet been adequately followed since his recent surgery and hospital discharge. One patient showed no improvement in hypertension, but thrombosis of the vein graft was subsequently proven by

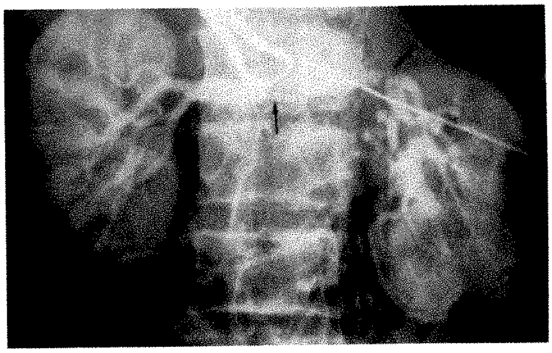

FIG. 7. Extremely severe bilateral renal artery stenosis in patient who has never been hypertensive.

arteriography which could explain the lack of response to surgery. Unfortunately, the patient refused nephrectomy of the infarcted kidney.

Seven patients with normal washout studies proved to have severe main renal artery stenoses on arteriography. These cases are considered false negative results, although split renal function studies performed on 4 were also normal. A fifth patient was said to have a positive Howard test performed elsewhere. In spite of the negative washout tests and normal split renal function studies, corrective surgery was performed on 4 of these patients on the basis of abnormal renograms and angiographic demonstration of renal artery stenosis. In none has there been significant improvement of hypertension.

DISCUSSION

During the past few years there has been a great enthusiasm for the diagnosis and surgical treatment of renovascular hypertension. Many patients have been cured, but also many have been subjected to surgery without improvement of their hypertension. Some patients have lost one kidney without cure and others have lost their lives.[2,7,14,17,20] It is now quite apparent that the selection of patients who will benefit from surgery is the most difficult and most important aspect of renovascular hypertension.

Basically, two different schools of thought can be found in the great amount of literature on this subject: (1) the anatomic approach, popular among radiologists and surgeons, which combines the patient's history of hypertension with positive angiographic findings,[14,16] and (2) the physiologic approach, claiming that renovascular hypertension is only curable if certain physiologic requirements are present, these being decreased sodium and water excretion and hyperconcentration of certain excreted substances as inulin, creatinine, etc.[4,11,21,27] It was our intention to approach this problem both anatomically, by intravenous pyelography and arteriography, and physiologically, by the urea washout test and split renal function studies. Since the excretion pattern of the modern diatrizoate contrast media follows closely that of inulin,[13] marked hyperconcentration of these substances in the ischemic kidney can be expected. Roentgenographically, this characteristic phenomenon is best demonstrated by a suddenly induced diuresis during intravenous peylography. Our still limited experience seems to suggest good correlation between hyperconcentration of creatinine, as determined during split renal function studies, and this modified intravenous pyelogram.

Good arteriography allows demonstration of all renal artery stenoses, but, contrary to common belief, visualization of such a lesion does not indicate curable renovascular hypertension. Visual estimation of the hemodynamic significance of a stenotic arterial lesion is very difficult unless a delayed blood flow or collaterals can be demonstrated. Obtaining pressure measurements across the area of narrowing during renal arteriography[2] requires technical skill and is not without its complications, as observed in 1 of our patients who developed a segmental postcatheterization thrombosis found at surgery. Furthermore, a patient may be found to have a considerable gradient and may not be cured by surgery. Contrary to animal ex-

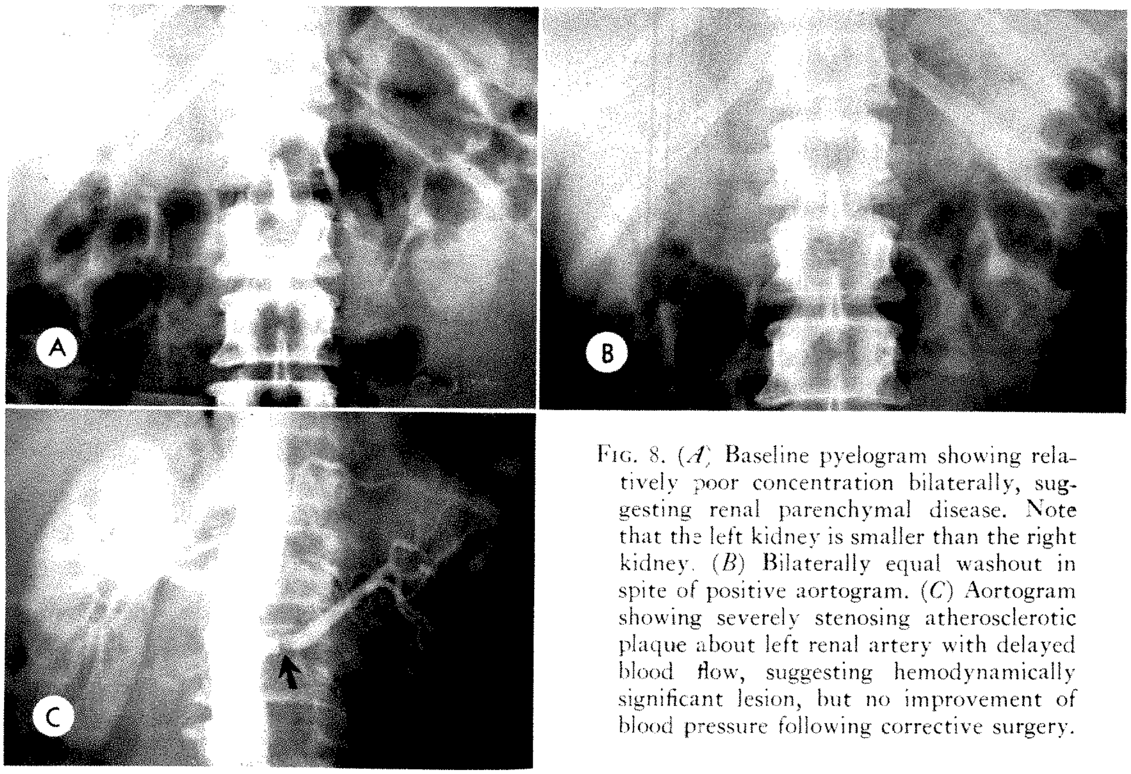

Fig. 8. (*A*) Baseline pyelogram showing relatively poor concentration bilaterally, suggesting renal parenchymal disease. Note that the left kidney is smaller than the right kidney. (*B*) Bilaterally equal washout in spite of positive aortogram. (*C*) Aortogram showing severely stenosing atherosclerotic plaque about left renal artery with delayed blood flow, suggesting hemodynamically significant lesion, but no improvement of blood pressure following corrective surgery.

perimental results, it is now well known[8] that severe narrowing of the renal artery in man does not necessarily result in hypertension (Fig. 7). In addition, hypertensive patients with hemodynamically significant renal artery stenoses, as proven by marked pressure gradients or by the presence of collaterals, may have normal split renal function studies and urea washout tests, while the radioactive renograms and arteriograms are abnormal (Fig. 8, *A*, *B* and *C;* and 9, *A*, *B* and *C*). Our experience from a few of these cases would indicate that such patients are not benefited by surgery. As more experience is gained, perhaps it will be found that all patients with unilateral renal artery stenoses and normal washout tests are better treated by medical means, rather than by unnecessary surgery. More informative data, including that obtained by renal biopsies, is necessary, however, and is being presently collected.

As a practical and easily performed procedure, the intravenous pyelogram with its modifications is believed to be the most important screening study for renovascular hypertension. In reviewing our cases, the diagnosis of renal artery stenosis could have been suspected on the rapid sequence intravenous pyelograms in 12 of the 14 proven positive cases. The findings, however, are often subtle and may escape less experienced observers. The washout test, on the other hand, emphasizes dramatically the difference in concentration and flow of urine and, most important of all, suggests surgically curable disease. The radioactive renogram, while capable of discovering more stenoses than does the washout test, is less specific, requires expensive equipment, experienced technicians and interpreters, and yields a high percentage of abnormal results.[24,25] Also, we have not found the radioactive renogram to be of prognostic significance, similar to the renal arteriogram. When combined with other studies, however, it should continue to serve as a useful method in screening and evaluating

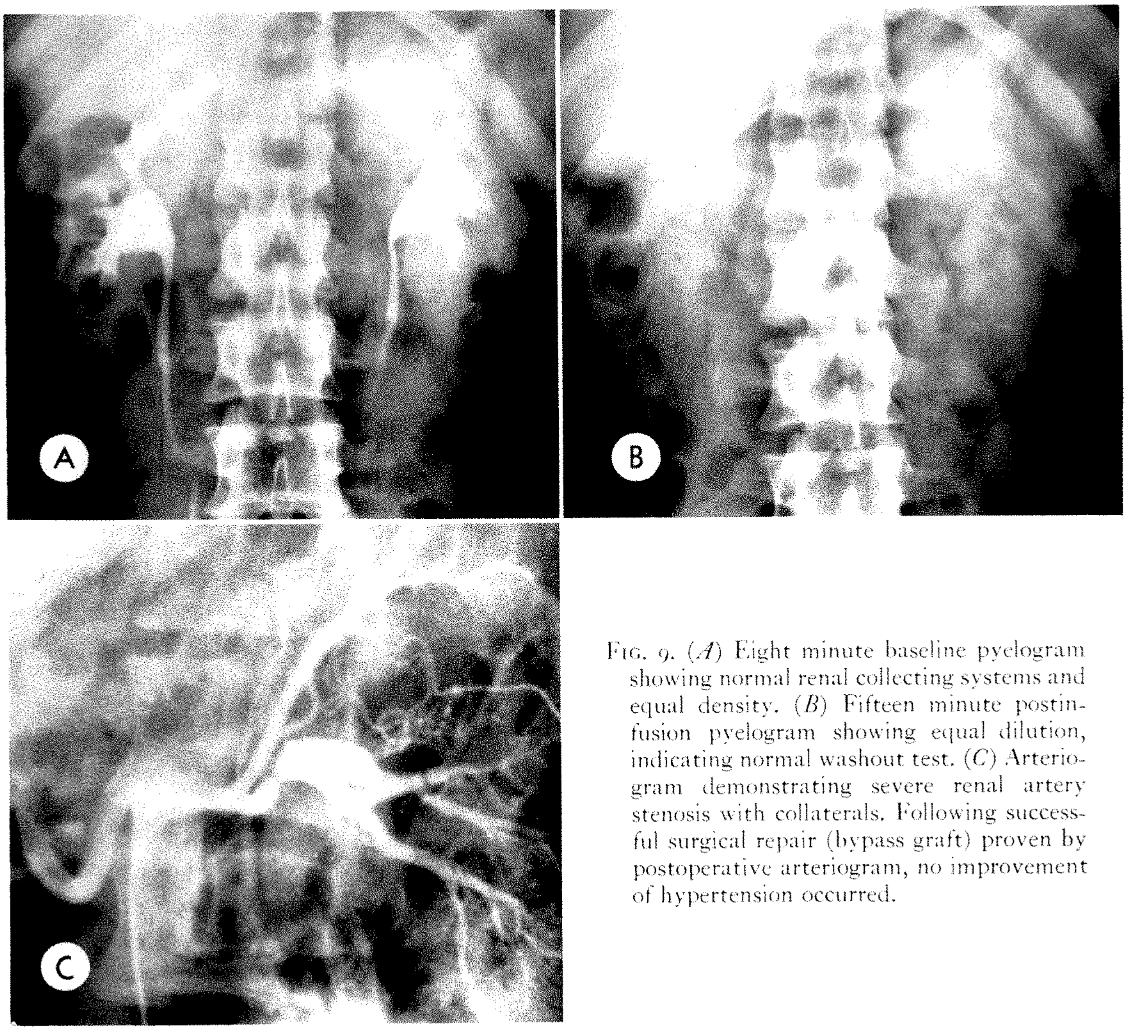

FIG. 9. (*A*) Eight minute baseline pyelogram showing normal renal collecting systems and equal density. (*B*) Fifteen minute postinfusion pyelogram showing equal dilution, indicating normal washout test. (*C*) Arteriogram demonstrating severe renal artery stenosis with collaterals. Following successful surgical repair (bypass graft) proven by postoperative arteriogram, no improvement of hypertension occurred.

hypertensive patients. While split renal function studies do provide important physiologic information of great prognostic significance, morbidity, technical difficulties and laboratory errors occur even in experienced hands.[6,14,18,25]

SUMMARY AND CONCLUSION

Intravenous pyelography with suddenly induced diuresis by a saline-urea infusion was peformed on 463 unselected hypertensive patients as a screening procedure without serious complications. The reliability of this test was correlated with that of the radioactive renography, renal arteriography and split renal function studies. In 3 per cent of the patients, the test was found to be positive. The cure rate in this group was excellent.

The urea washout test does not detect all renal artery stenoses, but surgical correction of such lesions, even if severe, very likely will not result in cure. In this respect the washout test is felt to be superior to renal arteriography and radioactive renography, since it involves selection of patients who may benefit from surgery. Consequently, it is felt that this examination shows promise as a simple roentgenographic screening procedure for the detection of surgically curable hypertension and warrants further trial. It is conceivable that it may fail in the rare patient with severe segmental renal artery stenosis, as may split renal function tests and radioactive renograms, and in those with equally severe bilateral disease, although no such case was found in our experience. Also, occasional difficulty in interpretation can be expected, particularly in cases with unilateral larger renal collection systems. Until more experience is gained, renal arteriography should be performed on all patients with strong clinical suspicion of renovascular hypertension to definitely exclude renal artery stenosis, regardless of the result of the urea washout test. Demonstration of a significant stenotic lesion together with the presence of a positive washout test is currently considered strong indication for surgery. Hypertensive patients with normal washout studies and angiographic demonstration of stenoses are probably better and more safely treated by antihypertensive drug therapy.

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Representatives on the American Board of Radiology: Justin J. Stein, Los Angeles, Calif.; Bernard P. Widmann, Philadelphia, Pa.; Donald S. Childs, Jr., Rochester, Minn.

Representatives on the National Committee on Radiation Protection: Robert Robbins, Philadelphia, Pa.; Herbert M. Parker, Richland, Wash.

Representative on the Board of Chancellors of the American College of Radiology: Milford D. Schulz.

Forty-seventh Annual Meeting: Roosevelt Hotel, New Orleans, La., April 8–10, 1965.

EDITORIAL

THE SIXTY-FIFTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Sixty-fifth Annual Meeting of the American Roentgen Ray Society was held at the Hotel Radisson in Minneapolis, Minnesota, from Tuesday, September 29 through Friday, October 2, 1964. President Ralph M. Caulk called the meeting to order and introduced John Paul Medelman, M.D., Chairman of the Executive Council, who in his capacity as President of the Minnesota State Medical Association voiced a cordial welcome to the Twin Cities.

Dr. Medelman presented interesting historic anecdotes of the Twenty-first Annual Meeting of the American Roentgen Ray Society which was held in Rochester and Minneapolis from September 14 to 17, 1920. The first day of that meeting was spent at the Mayo Clinic and—to quote Dr. Medelman—"The Pullman cars in which the members arrived were parked on the siding in Rochester so that it was unnecessary to remove the luggage. Toward evening the Society went by special train to Minneapolis, a three hour ride, where ample hotel accommodations were obtained." He also recalled that the Caldwell Lecture, which has become the most festive event of every Annual Meeting, was initiated at that meeting, "the brilliant success of this effort being tied up with the lusty and vigorous environment of its inception." A further note of interest was that on July 20, 1853, eleven enterprising young physicians met in the "fine new Court House of St. Paul, in the Minnesota Territory," and organized a medical society which was destined to become one of the most important in the land, as attested by the great Medical Anniversaries being celebrated today. This is the Centennial year of the Mayo Clinic; it was 75 years ago that the University of Minnesota Medical School opened its doors; and the Mayo Foundation for Medical Education and Research is now 50 years old. That Foundation is a unique alliance between a great public medical school and a world-famous private clinical and teaching facility.

"We are proud of our medical tradition in Minnesota," said Dr. Medelman in closing his eloquent Address of Welcome to the Society, "and as a State Medical Association we are happy that you have honored us by coming here."

President-Elect Harold O. Peterson, M.D., was then installed as President of the Society and was awarded the President's Medal by Dr. John Paul Medelman as Chairman of the Executive Council. Following a well-established tradition, the symbolic and working gavels were then presented to President Peterson.

Dr. Peterson's Inaugural Address, "First, a Radiologist," evoked considerable comment and characteristically stirred the imagination of the audience. In listening to this sometimes provocative Address, Dr. Peterson's fame as a teacher is easily understood. The Inaugural Address is published in full in this issue of the JOURNAL.

The Scientific Session of the Society began immediately thereafter with the presentation of 11 papers devoted to pediatric and neurologic roentgenology with an interesting paper on postoperative lymphocysts as a complication of pelvic lymphadenectomy. This first morning program was concluded by a discussion of the liver photoscan as an aid in the differential diagnosis of jaundice, and 2 papers dealing with cancerology.

The Scientific Exhibits were of high quality with the largest number on arteriography and vascular diseases. The scientific exhibits, assembled under the capable committee chairmanship of Dr. J. Scott Dunbar of Montreal, are described elsewhere in this issue of the Journal.

The Annual Caldwell Lecture was delivered by a renowned member radiologist, Dr. Richard Schatzki, on the subject of "Esophagus: Progress and Problems." Perhaps no one is better qualified in this area of gastrointestinal roentgenology than Dr. Schatzki, a fact attested by the long time designation of a structure of the distal esophagus as "Schatzki's ring." President Harold O. Peterson introduced Dr. Schatzki and at the end of his brilliant Lecture presented him with the Caldwell Medal.

On Wednesday the Scientific Session consisted of 9 papers with topics on visual and psychologic factors of roentgen interpretation; the application of ultrasonography to neurologic conditions; the application of thermography to various disease processes and several studies employing radioactive isotopes. All of these subjects indicate newer areas of interest in our discipline. The morning program was concluded by an excellent clinicopathologic study of Hodgkin's disease.

On Wednesday afternoon, in custom with previous years, the American College of Radiology presented a panel discussion with Dr. H. O. Peterson presiding, Dr. David S. Carroll, President of the American College of Radiology, Moderator, and Dr. Stanley M. Wyman, Dr. Robert W. McConnell, Dr. Wallace D. Buchanan, and Dr. Laurence L. Robbins as panelists on the subject, "Some Problems Confronting the American College of Radiology and the American Board of Radiology." This panel discussion created an unusually great interest and was attended by a large audience.

On Thursday morning the Scientific Session was comprised of papers dealing with laryngo-pharyngograms, pulmonary emphysema, pulmonary arteriovenous shunts, tension pneumothorax, gastric atrophy, intestinal necrosis, and intramural hemorrhage of the gastrointestinal tract, estimation of hepatomegaly and splenomegaly, 2 papers on radiation therapy and a more basic paper on radiation biology.

On Thursday evening, the Annual Banquet with the announcement of newly elected officers and presentation of Scientific Exhibit Awards proved most enjoyable. A unique stage show, featuring the celebrated Golden Strings and a balloon genius, was followed by a pleasant evening of dancing.

On Friday morning, 5 papers dealing with cardiovascular radiology, 1 on the study of metallic iron particles in the lymphatic and vascular systems, and 6 papers on arteriography completed the program.

The Director of Instruction Courses, Dr. Donn G. Mosser, as Dr. Peterson has in the past, collected a highly capable group of lecturers giving courses on various aspects of radiology. The number of technical exhibits assembled under the direction of our Honorary Member, Mr. Clifford L. Sherratt, and Manager of the Annual Meeting, Dr. James C. Cook, filled to capacity the hotel facilities. A list of technical exhibitors is presented elsewhere in the Journal.

The meeting was well attended and Minneapolis, the major commercial center of the north central Great Plains, was admired for her beauty and cleanliness.

The Committee on Local Arrangements under the Chairmanship of Dr. Cyrus Owen Hansen and the Ladies Committee under the Chairmanship of Mrs. Hansen organized interesting trips. The Mayo Clinic was visited on Monday, a trip attended by some of the gentlemen as well as ladies. Others played golf in the Annual

Willis F. Manges Tournament at the Hazeltine National Golf Club in rather brisk autumn air. A tour of Minneapolis was well attended on Tuesday, and on Wednesday a delightful luncheon at the North Star Inn included a spectacular historic millinery display, "Millinery Milestones." That evening the Tyrone Guthrie Theatre Party appreciated an excellent theatrical group in a most unusual theater. Before the Annual Banquet on Thursday, a choice was offered between a tour of the Minneapolis Institute of Arts and a sightseeing tour of the St. Paul and St. Croix River Valley. The Society is indebted to Mrs. Hansen and her Committee for a program of unequaled good taste and of variegated interest.

The Hotel Radisson provided excellent service throughout this meeting in every respect and the Society is appreciative of its efforts.

The Committee on Press Relations with Dr. Donn G. Mosser as Chairman was assisted by Mr. Otha W. Linton from the American College of Radiology.

Dr. Cyrus O. Hansen deserves special thanks for his capable management of local arrangements. The gratitude felt by the Society to Dr. John Paul Medelman for his warm welcome in the name of Minnesota medicine, for his past service to the Society and to the medical profession at large was appropriately expressed by his being made our next President-Elect.

President Peterson and Mrs. Peterson sparked the meeting with wit, warmth and friendliness, to make this another most enjoyable and successful Annual Meeting.

K. L. Krabbenhoft, M.D.

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THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Sixty-fifth Annual Meeting of the American Roentgen Ray Society held at the Radisson Hotel, Minneapolis, Minnesota, September 29 to October 2, 1964 were of unusually high standard and displayed in a most artistic fashion. They were located in the Gold Room, La Salle Suite, A, B and C and Radisson Suite A and B, in the immediate vicinity of the Grand Ballroom, Section 1, where all the Scientific Sessions were held. The great interest shown by the many visitors in viewing this extremely important visually documentative part of the meeting was surpassed only by the enthusiastic appraisal of the Scientific Awards Committee, which this year after thoughtful consideration has awarded a Gold Medal, Silver Medal, Bronze Medal and 13 Certificates of Merit.

The Society is greatly indebted and expresses its deep gratitude to Dr. J. Scott Dunbar and his untiring Scientific Exhibits Committee for assembling this magnificent collection of exhibits which contributed immeasurably to the success of the Sixty-fifth Annual Meeting.

The following is a description of the exhibits:

Gold Medal

Guided Angiography. Manuel Viamonte, Jr., M.D., and Mr. Robert C. Stevens, Research Consultant, University of Miami School of Medicine, Jackson Memorial Hospital, Miami, Florida.

During the last 3 years a new percutaneous technique for selective cardiac and vascular catheterizations has been developed by the exhibitors and applied to the study of numerous pathologic conditions. The basic elements of this new approach include: (1) a catheter with a deflector assembly which permits, in a unique fashion, full control of the angulation of the tip which is radiopaque, tapered and atraumatic; (2) extreme maneuverability of the catheter due to the special construction of its wall; and (3) a rotating cradle-type table top which can be adapted to any conventional roentgenologic unit and facilitates the fluoroscopic position of the catheter as well as the rotation of the patient during filming.

The applications of this technique are:

- (A) Morphologic and hemodynamic studies
  - (1) Cardiac and vascular pathology: congenital and acquired
  - (2) Mass lesions: tumor *vs.* inflammation
  - (3) Tumor: differentiating benign *vs.* malignant
- (B) Selective introduction of drugs

The technique used, practical demonstrations on a phantom model and examples of selective angiograms were presented in this beautiful exhibit.

The auxiliary equipment which consists of new guides, guide holders, trocar drapes and portable lead-barriers with a built-in shelf for the surgical and catheterization instruments was also shown.

Silver Medal

Intramural Hemorrhage in the Intestinal Tract. M. T. Khilnani, M.D., R. H. Marshak, M.D., Joan Eliasoph, M.D., and B. S. Wolf, M.D., The Mount Sinai Hospital, New York, New York.

Intramural hemorrhage of the small bowel and colon as a complication of hemorrhagic diathesis and anticoagulant therapy is being recognized with increasing frequency. Roentgen study has revealed a characteristic pattern in many of these cases. Recognition of this pattern and differentiation from other discrete lesions of the intestinal tract are important since operation is unnecessary and resolution the rule.

The roentgen changes were studied in a variety of patients with hemorrhagic diathesis and in patients bleeding from anticoagulant therapy.

Differential diagnosis from mesenteric vascular occlusion and other disorders was described and authoritatively illustrated.

Bronze Medal

Chemotherapy Combined with Irradiation in the Treatment of Squamous Cell Carcinoma of the Head and Neck. John F. Daly, M.D., and Milton Friedman, M.D., New York University Schools of Medicine, New York, New York.

To determine whether chemotherapy could enhance radiation effects, a controlled clinical study was conducted on more than 100 patients with squamous cell carcinomas of the head and neck, which were treated with irradiation and oral methotrexate. The two types of interior controls were: (1) methotrexate plus a sublethal dose of radiation, and (2) in the presence of multiple metastatic lymph nodes, each one was treated with a different combination of the two modalities.

Since any consideration of the effect of combined therapy on cure rates would be premature, only the radiobiologic effect on the treated tumor was analyzed in this strictly documentative exhibit.

Ten lesions, arrested for 7 to 24 months, comprised the chief evidence that methotrexate enhanced radiation effects. One other type of interior control, based on multiple metastatic lymph nodes, also supported the thesis of enhancement.

The visitors of this exhibit were provided with an artistically prepared guide booklet which carried on its front cover a remarkable reproduction from Quaderni d'Anatomia by Leonardo da Vinci (1452–1519).

Certificates of Merit

1. *Selective Celiac and Superior Mesenteric Arteriography.* Stanley Baum, M.D., Moreye Nusbaum, M.D., Arthur Finkelstein, M.D., and William Blakemore, M.D., Graduate Hospital University of Pennsylvania, Philadelphia, Pennsylvania.

Selective celiac and superior mesenteric arteriograms were shown illustrating the diagnostic application of the examination in gastrointestinal disorders.

Emphasis was placed on the high incidence of anatomic variations as well as on the value of stereoscopic filming.

2. *Rheumatoid Arthritis of Hands and Feet; Roentgen Changes, Surgical Treatment.* David L. Berens, M.D., P. A. Casagrande, M.D., L. Maxwell Lockie, M.D., and Bernard M. Norcross, M.D., Buffalo General Hospital, Buffalo, New York.

The clinical, laboratory and roentgenographic changes were correlated with the surgical and pathologic findings.

The *early* roentgen features were demonstrated.

The role of synovectomy and other surgical procedures was stressed.

The pathogenesis of erosions and cysts was illustrated.

3. *Acro-Osteolysis.* William D. Cheney, M.D., and J. Clyde Spencer, M.D., Edward W. Sparrow Hospital, Lansing, Michigan.

Familial acro-osteolysis is a rare and bizarre disease of dominant inheritance pattern.

The exhibit comprised roentgenograms of 5 members of a single family demonstrating nonreactive osteolytic destruction of phalanges, platybasia, wormian bone formation, dolichocephaly in the male, premature suture closure in the female, atrophy of mandibular and maxillary alveolar margins, and early edentulism.

Demineralization of the bones, particularly the vertebrae, appeared to correlate with decreasing height as the disease progressed.

4. *Arteriography in the Community Hospital.* Joseph L. Curry, M.D., and Willard J. Howland, M.D., Ohio Valley General Hospital, Wheeling, West Virginia.

This exhibit was based upon experience in performing over 500 arteriographies in over 400 patients during the last 2 years.

Emphasis was placed upon minimum essential materials and equipment required, as well as the technical approaches which render these procedures efficient and safe at the community hospital level. The ability to use various technical approaches was discussed as a prerequisite for a high degree of success.

The responsibility of the community radiologist to investigate and report his observations was stressed.

5. *Micturition Cysto-Urethrography by the Excretory Method in Infancy and Childhood.* J. S. Dunbar, M.D., M. B. Nogrady, M.D., and B. St. J. Brown, M.D., The Montreal Children's Hospital, Montreal, P.Q., Canada.

The method of examination was explained briefly and illustrated with transparencies and 16 mm. cinefluorograms.

Normal and abnormal examples of micturition cysto-urethrograms in infancy and childhood (few days old to 14 years) were shown, obtained by the excretory method (transparencies and cinefluorograms).

The automatic exposure device (P-O-Mat) used at The Montreal Children's Hospital was shown *in toto*, illustrating the positioning of the patient and the attachment of the perineal unit (male and female).

Evaluation of the method was given, based on 1,000 urographies.

The following conclusions were reached: Study of the lower urinary tract by voiding cysto-urethrography can be done by utilizing intravenously administered contrast medium in a majority of pediatric patients unless there is severe reduction of renal function. Each examination should be planned individually. This study prolongs considerably the time required for excretory urography, but the results justify the additional time and effort. In infants, the bladder fills and empties so frequently that dilution of contrast medium is not a problem. In children, the most important factor in achieving successful lower tract visualization is to have the bladder empty before the injection is made, thus avoiding dilution of opaque medium in the bladder.

The main advantages include: Minimal instrumentation; full study of the urinary tract in one visit and diagnostic information obtained under more physiologic conditions.

6. *Selective Renal Arteriography in Medical Diseases of the Kidney.* R. S. Foster, M.D., W. H. Shuford, M.D., E. P. Tuttle, M.D., and H. S. Weens, M.D., Emory University School of Medicine, Atlanta, Georgia.

This exhibit highlighted the fact that there are angiographically distinguishable lesions of the small renal arteries not generally recognized by abdominal aortography, and that these lesions fit certain patterns which have been helpful in differentiating primary vascular from parenchymal disease of the kidney.

Selective renal arteriograms of benign and malignant hypertension, pyelonephritis, tuberculosis, hydronephrosis, renal artery occlusion, cystic disease of the kidney and malignant tumors were shown.

7. *Total Body Opacification.* N. Thorne Griscom, M.D., and E. B. D. Neuhauser, M.D., Children's Hospital Medical Center, Boston, Massachusetts.

As contrast material mixes with the blood (following injection for intravenous urography, for example), the entire circulation becomes slightly opaque. The added opacity of an organ is proportional to the amount of blood it contains. An avascular lesion, however, will keep its former density and will sometimes be seen as an area of lucency, contrasting with the newly opacified structures around it. This effect has been useful in the demonstration of cysts, cystic and necrotic tumors, severe hydronephroses, renal infarctions, hematomas, abscesses, ascites, thrombosed hemangiomas, and volvuli.

Examples of the effect in abdominal lesions in infancy were shown.

8. *The Keratosulfate Mucopolysaccharidosis of Morquio (Morquio's Disease); Roentgenographic Features of a Chemically Defined Disease Entity.* Leonard O. Langer, Jr., M.D., and John A. Tobin, M.D., University of Minnesota Hospitals, Minneapolis, Minnesota.

Morquio and Brailsford independently

described this dwarf condition in 1929. It is commonly known as Morquio's disease or Morquio-Brailsford chondro-osteodystrophy. Lately, the term Morquio-Ullrich syndrome has been applied to the condition.

Recent chemical investigation has revealed an abnormal mucopolysaccharide, keratosulfate, in the urine of individuals with Morquio's disease. This is a similar substance to that found in the urine of individuals with the Hurler syndrome. The roentgenologic features appear characteristic by the time clinical abnormality is noted. These features were illustrated in the infant, child, and adult.

9. *The Pattern of Bone Erosion in the Hand and Wrist in Rheumatoid Arthritis.* William Martel, M.D., John T. Hayes, M.D., and Ivan F. Duff, M.D., The University of Michigan, Ann Arbor, Michigan.

The bone erosions in the hand and wrist in rheumatoid arthritis assume a characteristic and predictable pattern which correlates closely with anatomic and mechanical factors. This pattern was described and illustrated.

10. *Tomography with "Mimer."* Ove Mattsson, M.D., Serafimer Hospital, Stockholm K, Sweden.

Tomography with "Mimer" can be performed using both linear and circular motion. The linear tomography can be carried out in any position of the plane between horizontal and vertical. The circular tomography, which uses 180° of a full circle, is designed for the horizontal plane.

Series of tomograms were shown demonstrating applications in various fields (encephalography, examinations of the chest, skull, bile ducts) as well as phantom tests. The latter verified the high stability of the mechanical system, which may explain the good results obtained.

11. *Subselective Gastrointestinal Angiography.* Robert E. Paul, Jr., M.D., Harry H. Miller, M.D., Paul C. Kahn, M.D., James F. Patterson, M.D., Allen D. Callow, M.D., Alice Ettinger, M.D., and Ralph A. Deterling, Jr., M.D., Tufts-New England Medical Center—New England Center Hospital, Boston, Massachusetts.

Application of percutaneous catheterization of gastrointestinal vessels, including secondary and tertiary branches, and demonstration of this technique for surgical and chemotherapeutic management of malignancies formed the theme of this exhibit.

Normal vasculature and tumor vessels involving liver, pancreas and gastrointestinal tract were illustrated.

12. *Detection of Occult Metastatic Cancer in Bone by Scanning with Radioactive Strontium 85.* David M. Sklaroff, M.D., N. David Charkes, M.D., and J. Gershon-Cohen, M.D., D.Sc. (Med.), Albert Einstein Medical Center, Northern Division, Philadelphia, Pennsylvania.

Photoscanning with strontium 85 was presented as a method of diagnosing occult metastases of cancer in bone. Representative roentgenograms, photoscans, and histopathologic photographs were shown. The results obtained in the study of 130 patients were analyzed and correlated with the roentgenologic and pathologic findings.

13. *Staging of Bladder Cancer; A New Method Using a Triple Contrast Technique.* Donald A. Taylor, M.D., Kevin L. Macken, M.D., and Arnold L. Bachman, M.D., Institute for Cancer Research, College of Physicians and Surgeons, Columbia University-Francis Delafield Hospital, New York, New York.

An amplification of the double contrast method of evaluating bladder tumors (arteriography plus intravesical gas) was presented.

Formerly, the simple double contrast method was used by the exhibitors and found to be grossly inadequate for the accurate pretreatment staging of bladder neoplasms. The inadequacy was due to inability to visualize the spread of the neoplasm into the perivesical area. Now they have added to the double contrast method the injection of perivesical gas, thus affording accurate delineation of the entire tumor and its blood supply.

The correlated findings in 20 cases so studied were discussed and illustrated.

OTHER EXCELLENT EXHIBITS WERE:

Extremity Cylinder-Volume Ratio for Evaluating Diseases in Infants and Children. D. H. Altman, M.D., and R. E. Litt, M.D., Variety Children's Hospital, Miami, Florida.

In the pediatric age group, a relatively constant relationship between the muscle mass and total cylinder diameters of the extremities has been noted. These ratios have been determined by analysis of hundreds of cases originally studied for leg length determination; measurements have been taken at mid-femur and mid-tibia. In normal infants, this ratio is between 0.64 and 0.72.

Alteration of this ratio should alert the radiologist to the diagnosis of a specific disease group affecting soft tissues and/or muscle tissue either primarily or secondarily. Disorders altering the normal ratio were presented.

Stereoscopy. Joshua A. Becker, M.D., George L. Popky, M.D., and Herbert M. Stauffer, M.D., Temple University Hospital, Philadelphia, Pennsylvania.

The place of stereoscopy in the practice of radiology is still debated, although stereoscopic roentgenograms were produced early in the development of radiology.

The exhibitors feel that in specific instances the stereoscopic technique is valuable, and is not replaceable by oblique projections. The reluctance of radiologists to use a three-dimensional examination may be the result of poor stereoscopic vision.

The exhibit presented both a test for stereoscopic vision and examples of the value of the stereoscopic examination.

New Technique for Tumor Localization Utilizing Iodinated Serum Albumin and Regional Oxygenation. R. E. Collier, M.D., J. W. Finney, M.A., G. A. Balla, M.D., and J. T. Mallams, M.D., Charles A. Sammons Research Division, Baylor University Medical Center, Dallas, Texas.

This exhibit described a new technique, developed by the exhibitors, utilizing iodinated serum albumin in combination with regional oxygenation to demonstrate tumors not demonstrable previously by isotope techniques. Control animal studies employing VX-2 tumors were illustrated.

The panels showed the technique and representative scans outlining various human tumors including liposarcoma, fibrosarcoma, squamous cell carcinoma of the antrum, metastatic adenocarcinoma of the liver from the colon, and squamous cell carcinoma of the cervix.

Echoencephalography. J. Gershon-Cohen, M.D., D.Sc. (Med.), and G. Elizondo-Martel, M.D., Albert Einstein Medical Center, Northern Division, Philadelphia, Pennsylvania.

When an ultrasonic beam of appropriate frequency is directed through the skull, the echo reflected by the mid-line structures of the brain can be recorded.

The position of this echo indicates the position of the midline structures and indirectly demonstrates the presence of space-occupying lesions. Thus, any condition that tends to displace the midline structures will displace the position of the echo. Even cerebro-atrophic processes can be detected by measuring the 3rd ventricle width, while in hydrocephalus, it is possible to determine the thickness of the remaining cerebral layer or the expanded inferior horn of the lateral ventricle.

Among 170 patients, including normals and those with tumors, metastases, subdural hematomas and thrombosis, echo shift was present in all space-occupying lesions.

Echoencephalography is atraumatic, rapid, and free of discomfort for the patient and shows promise of becoming another indispensable diagnostic discipline. Its possible implications were demonstrated in the exhibit.

The Isotope Encephalogram. J. K. Goodrich, M.D., Forrest T. Tutor, M.D., and

Ottis G. Ball, M.D., University of Mississippi Medical Center, Jackson, Mississippi.

A review of 350 consecutive brain scans revealed 83 diagnosed brain tumors of which 71 had positive brain scans.

This exhibit reported the correlation of brain scan findings with a diagnostic battery consisting of isotope encephalography, neurologic physical examination, skull roentgenography, carotid arteriography, pneumoencephalography, and ventriculography.

The brain scan maintained a high degree of accuracy and was felt to have proved itself as an excellent first step procedure in screening neurologic patients. To this end its advantages are multiple for there is no reported patient morbidity, no postprocedural sequelae, and it lends itself well to outpatient examination.

Clinical Applications of Thermography. J. D. Haberman, M.D., Eric Brueschke, M.D., and J. Gershon-Cohen, M.D., Sc.D. (Med.), Albert Einstein Medical Center, Northern Division, Philadelphia, Pennsylvania.

During the past 3 years the exhibitors were interested in thermography. They were exploring its diagnostic potentialities in diseases of the breast, localization of the placenta, its place in the prognosis of arthritis, peripheral vascular disease, and in the detection of primary and secondary sites of malignancy.

The highlights of thermography were shown in all of these conditions.

Arteriography in Orthopedic Disease. Mordecai Halpern, M.D., and Robert H. Freiberger, M.D., The New York Hospital—Cornell Medical Center, New York, New York.

This exhibit presented examples of the applications of arteriography in orthopedic disease, to obtain a more precise understanding of various morbid conditions and to determine its preoperative value as a therapeutic guide. Arteriograms of benign and malignant bone and soft tissue tumors were shown as well as of the investigations of the scalenus anticus syndrome and posttraumatic vascular conditions.

The indications, contraindications and limitations of this diagnostic modality were discussed.

Intravenous Abdominal Aortography. Samuel D Hemley, M.D., Richard D. Kittredge, M.D., Virginia Kanick, M.D., and Nathaniel Finby, M.D., St. Luke's Hospital, New York City, New York.

The exhibitors presented their technique of intravenous abdominal aortography. Cases depicting its use in nephrotomography, dissecting aneurysms, arteriosclerotic aneurysmal disease, peripheral vascular disease, etc., were demonstrated.

Arteriographic Demonstration of Bladder Tumors. E. K. Lang, M.D., William Niles Wishard, M.D., John W. Beeler, M.D., and Myron Nourse, M.D., Methodist Hospital, Indianapolis, Indiana.

The arteriographic demonstration of bladder tumors allows accurate staging according to the Jewett classification. Thus a preoperative assessment of the tumor is possible. The histologic differentiation of the tumor is often suggested by the type of stain seen. The method affords excellent follow-up examinations after cobalt 60 irradiation.

The exhibit presented some unusual examples documenting the value of the method.

Radon and Radioactive Seed Volume Implants for Extensive Recurrent Vaginal-Pelvic Cancer. Leonard M. Liegner, M.D., St. Luke's Hospital, New York, New York.

Thirteen cases were presented in which extensive recurrent vaginal-pelvic cancers were treated by radon and radioactive seed interstitial volume implants. The results of such treatment were detailed. Specifically charted were the degree of tumor regression, tumor control, the extent of prolongation of life, and both immediate and late tissue reactions or complications.

The various interstitial techniques were pictorially shown as well as "mock ups" of the actual equipment used for the three recommended methods; *i.e.*, (1) permanent seed implants, (2) "blind end" removable iridium 192 nylon ribbons, and (3) removable ribbon afterloading. Roentgenograms and histories of representative cases were included.

Segmental Occlusion of the Middle Cerebral Artery. Leon Love, M.D., Cook County Hospital, Chicago Medical School, Sanford J. Larson, M.D., Marquette University Medical School, and Charles F. Timmons, M.D., Cook County Hospital, Chicago, Illinois.

This exhibit presented 42 cases of segmental occlusion of the middle cerebral artery which had been diagnosed by carotid arteriography. The ascending frontoparietal and sylvian branches were segmentally occluded and serial roentgenograms demonstrated collaterals to the area.

Changes Due to Trauma in the Fat Overlying the Pronator Quadratus Muscle; A Roentgenologic Sign. Douglas W. MacEwan, M.D., and D. J. Sieniewicz, M.D., The Montreal Children's Hospital and The Montreal General Hospital, Montreal, P.Q., Canada.

A linear fat plane overlying the pronator quadratus muscle is altered in roentgenologic appearance following trauma or as a result of inflammation. The exhibit demonstrated the particular usefulness of this sign in undisplaced fractures of the lower ends of the radius and ulna which might otherwise be overlooked.

Newer Concepts of Abdominal and Pelvic Arteriography. Thomas R. Marshall, M.D., and J. T. Ling, M.D., University of Louisville School of Medicine, Louisville, Kentucky.

Newer concepts of abdominal and pelvic arteriography were discussed. The exhibitors' experience in over 600 brachial and axillary injections (non-catheter) and 200 transfemoral injections (Seldinger technique) was described.

The brachial artery or axillary technique is preferred for the majority of conditions, and especially in patients over 50 years of age or persons with vascular disease of the iliac and femoral vessels.

A Template for the Localization of the Head of the Newborn Femur; An Aid in the Early Diagnosis of Congenital Dislocation of the Hip. Arthur L. Matles, M.D., New York Medical College—Flower Fifth Avenue Hospital, New York City, New York.

Demonstration of a transparent template which accurately localizes the cartilaginous head of the femur on the roentgenogram of a newborn up to 4 weeks of age was the subject of this exhibit. The template has been used on roentgenograms of 50 stillborns with arthrograms and 110 newborns with suspected congenital dislocation of the hip. There was excellent correlation in 98 per cent of the cases.

Gastrointestinal Mucosal Studies with a New Contrast Medium. Ove Mattsson, M.D., Serafimer Hospital, Stockholm K, Sweden.

The delineation of the mucosal relief in gastrointestinal examinations can be considerably improved by using a new special contrast medium in which the stabilization of the heavy barium sulfate is obtained without an increase of viscosity. The low-viscosity barium meal seems to adhere more closely to the mucosa, thus producing greater details in the mucosal relief.

Roentgenograms from various parts of the gastrointestinal tract were displayed.

The development of the new preparation was made in cooperation with Mr. G. Embring, Upsala, Sweden.

Brain Scanning in Non-Tumorous Conditions. Samuel B. McCarter, M.D., David D. Beiler, M.D., and John L. Williams, M.D., Ph.D., Geisinger Medical Center, Danville, Pennsylvania.

It is now well known that many conditions other than tumors may produce abnormalities on brain scans. This exhibit illustrated most of these. In addition, it included serial scans (Hg^{197}) in some, chiefly vascular accidents, to show the times at which one can expect the scan to first become positive, to reach its maximum intensity and finally to return to negative.

The Diagnostic Value of Selective Renal Arteriography. Fleming McConnell, M.D., F.R.C.P. (C), and Joseph Kiss, M.D., The Montreal General Hospital, Montreal, P.Q., Canada.

Selective arteriography of the kidneys has created a new field of roentgenologic diagnostic interpretation. The particular value of the examination is in expanding processes of the kidney, and cases illustrating the classic and the less classic arteriographic appearances were shown. In addition, the value of the examination in other conditions, including hydronephrosis, renal trauma and renal vascular occlusion, was demonstrated.

The exhibit was selected from a series of 90 selective arteriograms including 28 patients with a renal mass.

Neurofibromatosis. William Meszaros, M.D., Hildegarde Schorsch, M.D., and Francis Guzzo, M.D., Cook County Hospital, Chicago, Illinois.

Although many pathologic aspects of neurofibromatosis are still poorly understood; the roentgen patterns are frequently characteristic. Knowledge of the many manifestations of neurofibromatosis enables the radiologist to assist in the diagnosis of this condition, especially when the clinical picture is not typical. Examples of such manifestations were shown.

The American College of Radiology Foundation Museum (National Council of Radiological Heritage). Robert T. Morrison, George Eastman House, Rochester, New York.

The types and genera of radiologic memorabilia needed for the permanent collection of the Museum had token representation in this exhibit. By discussion, the aims and purposes of the program were presented. The procedures of donation and instructions for delivery were made available in printed form.

Intra-Osseous Lumbar Vertebral Venography: A Diagnostic Aid in Diskogenic Disease. Ernest J. Pick, M.D., Murray R. Glickman, M.D., Constantin Cope, M.D., and J. Gershon-Cohen, M.D., Albert Einstein Medical Center, Northern Division, Philadelphia, Pennsylvania.

The technique of intra-osseous vertebral venography was presented by means of a series of color transparencies. These depicted the successive steps in the performance of the procedure. Diagrams illustrated the normal anatomy of the vertebral venous plexuses. Normal intraosseous vertebral venograms, along with schematic drawings, preceded the presentation of 4 cases of special interest. The studies were analyzed in detail.

A Comparative Study of Three Cleansing Agents for Bowel Visualization in Roentgenography. Jirair N. Sarian, M.D., Blaine Sarian X-Ray Laboratories, Los Angeles, California.

Some salient aspects of the problems of bowel visualization in roentgenography and a comparative study of visualization achieved with 3 commonly-employed cleansing agents in various roentgenographic procedures involving the gastrointestinal tract were presented.

The 3 cleansing agents were: (1) standardized senna concentrate known under the trade name of X-Prep Powder ($\frac{3}{4}$ oz. dose, oral, 16 hours before the scheduled procedure); (2) preparation of sodium biphosphate-sodium phosphate (2 to $2\frac{1}{2}$ oz. dose, oral, 12 hours before procedure); and (3) castor oil (2 to $2\frac{1}{2}$ oz. dose, oral, 16 hours before scheduled procedure).

The following conclusions were reached: (a) standardized senna concentrate resulted in significantly more effective visualization than the sodium biphosphate-sodium phosphate preparation and both were distinctly more efficacious than castor oil; (b) the advantages of standardized senna concentrate were considerably enhanced by the simplicity of oral administration, the substantially lower incidence of side effects, the relatively greater comfort to the patient, and the palatability of the powder; and, therefore, (c) it appeared to possess the criteria for an effective bowel evacuant preparatory to barium enema and intravenous pyelographic procedures.

Stress Fractures. Captain Bernard Schencker, M.D., and Captain David L. Moody, M.D., U. S. Army Hospital, Fort Jackson, South Carolina.

Early detection and proper evaluation of stress fractures depend on familiarity with the characteristic clinical features and, importantly, the variable roentgenographic appearance.

Roentgenographically, stress fractures may be conveniently classified into 5 different categories: *Linear Type:* There is a linear radiolucent cleft extending obliquely through the cortex of the bone. This varies from a fine line to a well defined defect, which at times extends across the entire shaft of the bone. *Periosteal Type:* A fracture line is not visible. A small "puff" of periosteal reaction demarcates the lesion. Later, the periosteal reaction enlarges and dense callus is formed. *Sclerotic Type:* An area of endosteal sclerosis of varying density, configuration and size defines this type, which occurs in cancellous bone. Notable examples include the os calcis, the tibial metaphysis and the proximal first metatarsal. *Fragmentary Type:* This type is almost always confined to the metatarsals and resembles a comminuted crush-type fracture. It is less common than the other types. *Mixed Type:* Any of the four basic types may occur simultaneously or may appear at some stage in the evolution of a lesion. When two or more types are present, the fracture is classified as mixed. Most lesions show mixed features.

Examples of each type were presented and the pertinent data discussed in detail.

Drip Infusion Pyelography and Nephrotomography. Captain Bernard Schencker, M.D., and Colonel Peter Zanca, M.D., Brooke General Hospital, BAMC, Fort Sam Houston, Texas.

Drip infusion of dilute contrast material has proven to be a simple, safe and reliable technique for obtaining high quality pyelograms and diagnostic nephrotomograms.

The techniques of drip infusion and nephrotomography were detailed and interesting illustrative cases were presented.

Bone Lesions in Sarcoidosis. Hildegarde Schorsch, M.D., Gertrude Engbring, M.D., Bernice Perdziak, M.D., and Hyo Hyun Byun, M.D., Cook County Hospital, Stritch School of Medicine, Chicago, Illinois.

Various types of bone lesions in sarcoidosis with long term observations were demonstrated in this exhibit.

Genitography in Intersexual States. Charles E. Shopfner, M.D., Children's Memorial Hospital-University of Oklahoma Medical Center, Oklahoma City, Oklahoma.

An intersex state exists where there are contradictions in the morphology of the genital tract. A practical sex should be promptly assigned to infants with an intersex problem so that the sex of rearing will ensure proper gender role.

Genitography provides knowledge of the internal genital passages which is important in the assignment of a practical sex. The technical aspects of genitography were discussed and a classification of genitography findings was presented. Proper usage of genitography in conjunction with other diagnostic methods will ensure a genital life as near the anatomic capabilities as possible.

Craniolacunia (Lückenschädel). Charles E. Shopfner, M.D., J. T. Jabbour, M.D., and R. M. Vallion, M.D., Children's Memorial Hospital-University of Oklahoma Medical Center, Oklahoma City, Oklahoma.

Craniolacunia has been observed in 90 of 177 patients with meningomyelocele and/or anencephalocele who had skull roentgenograms during the first 3 months of life.

Information concerning incidence, relation to neural arch anomalies and hydrocephalus, course and prognosis of craniolacunia was presented. Case material was used to illustrate a proposed classification of cranial bone defects. The roentgen findings of craniolacunia and the differential diagnosis were described. The etiology and significance were briefly discussed.

Selective Aortography in Diagnosis of Aortic Valvular Disease. Harry L. Stein, M.D., Mordecai Halpern, M.D., and John A. Evans, M.D., The New York Hospital—Cornell Medical Center, New York, New York.

Catheter aortography was used as a means of diagnosis and evaluation of aortic valvular disease in 83 patients.

In this exhibit specific information regarding the size of the aortic root and ascending aorta, degree of valvular stenosis or insufficiency, status of the coronary arteries and sinuses of Valsalva was presented. The data obtained were correlated with physiologic data, such as the gradients across the aortic valves and size of valve orifice, and the findings at surgery. In several patients angiographic re-evaluation of the aortic valves or valve protheses following surgery was secured and the results were shown.

The percutaneous right transaxillary route is considered to be the best approach for these studies.

Congenitally Abnormal Feet. Arch W. Templeton, M.D., University of Missouri School of Medicine, Columbia, Missouri, William H. McAlister, M.D., and Irwin D. Zim, M.D., The Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

The terms "clubfoot" and "talipes" have become meaningless. This exhibit aimed to standardize the terms employed to describe congenital foot abnormalities.

The basic positional deformities of the foot were analyzed with line drawings and roentgenograms employing the proposed terminology. The fundamental roentgenologic technical principles were outlined. The normal range of angles formed by different bones of the feet was determined and the diagnostic significance evaluated roentgenologically.

The Callosal Sulcus Sign; A Valuable Sign in Failed Pneumoencephalograms Due to Posterior Fossa Tumors and Other Obstructing Lesions. Alan E. Zimmer, M.D., Harold G. Jacobson, M.D., and Mannie M. Schechter, M.D., Montefiore Hospital and Albert Einstein College of Medicine, New York, New York.

Althouth failure to fill the ventricular system by lumbar pneumoencephalography may result from physicomechanical factors of doubtful clinical significance, posterior fossa tumors and other obstructing lesions may likewise impair normal ventricular filling.

Examples were shown of more than 20 such cases in which air demonstrated a dilated sulcus of the corpus callosum tending to confirm the presence of tumor or other obstructing lesions. Alterations in size and shape of the subarachnoid cisterns aided greatly in the roentgen diagnosis and examples of these were also shown.

THE TECHNICAL EXHIBITS

THE TECHNICAL EXHIBITS of the Sixty-fifth Annual Meeting of the American Roentgen Ray Society held at the Hotel Radisson, Minneapolis, Minnesota, September 29 to October 2, 1964, were located in Section 2 and 3 of the Grand Ball Room, in the immediate vicinity of the Scientific Sessions. This arrangement, together with the fact that President-Elect Harold O. Peterson and his Committee programmed all Scientific Sessions in the morning, provided the members of the Society and their guests with ample time to visit the Exhibits which were of exceptional quality and most attractive.

Mr. Clifford L. Sherratt, Honorary Member and Dr. James C. Cook, Manager of the Annual Meeting spared no time or effort in making the necessary preparations and a description of the Exhibits was provided in the Section of Technical Exhibits of the Program Booklet which was presented to every registrant of the Annual Meeting. A preliminary perusal of these descriptions was of great aid to the unusually large number of visitors in appraising the many splendid technical advances made recently in every branch of radiology. The displays included a dazzling array of new devices; instruments; diagnostic and therapeutic apparatus; cine and television viewing systems; films, intensifying screens and processing chemicals; contrast media; great books, monographs and other publications; a diagnostic ultrasonoscope; radioactive pharmaceuticals; the latest development in projectors; and many other important items of timely interest to the radiologist.

The Society expresses its sincere thanks and profound appreciation to the exhibiting firms who contributed so importantly to the great success of the Sixty-fifth Annual Meeting.

The following is a list of the firms which participated in the Technical Exhibits:

Abbott Laboratories, North Chicago, Illinois; Albert-Acan X-Ray Solutions, Inc., Detroit, Michigan; Ames Company, Inc., and Ames Atomium, Inc., Elkhart, Indiana; Ansco X-ray Products, General Aniline & Film Corporation, Binghamton, New York; The Automatic Seriograph Corporation, College Park, Maryland; Bar Ray Products, Inc., Brooklyn, New York; Barnes-Hind Barium Products, Sunnyvale, California; Bell-Craig, Inc., New York, New York; Cenco-X-Ray Company, Chicago, Illinois; Coreco Research Corporation, New York, New York; Cordis Corporation, Miami, Florida; Dunlee Corporation, Bellwood, Illinois; E. I. du Pont de Nemours & Company, Inc., Wilmington, Delaware; Dynalectron Corporation, Mattern Division, Lake City, South Carolina; Eastman Kodak Company, Rochester, New York; Eureka X-Ray Tube Corporation, Chicago, Illinois; E-Z-Em Company, Inc., Port Washington, Long Island, New York; Forsyth X-Ray Corporation, Rochelle, Illinois; E. Fougera & Company, Inc., Hicksville, Long Island, New York; Franklin X-Ray Corporation, Philadelphia, Pennsylvania; General Electric Company, Milwaukee, Wisconsin; The Gevaert Company of America, Inc., Teterboro, New Jersey; Gordon Consultants, New York, New York; Great Books of the Western World, Chicago, Illinois; Grune & Stratton, Inc., New York, New York; Halsey X-Ray Products, Inc., Brooklyn, New York; Hoeber Medical Division, Harper & Row Publishers, Inc., New York, New York; Howdon Videx Products Corporation, Mount Vernon, New York; Ilford Inc., New York, New York; Keleket, Division of Laboratory for Electronics, Inc., Waltham, Massachusetts: Liebel-Flarsheim Company, Cincinnati, Ohio; The Machlett Laboratories, Inc., Springdale, Connecticut; Mallinckrodt Pharmaceuticals, St. Louis, Missouri; Micro X-Ray Recorder, Inc., Chicago, Illinois; Motorola Communications and Electronics, Inc., Chicago, Illinois; North American Philips Company, Inc., New York, New York; Pako Corporation, Minneapolis, Minnesota; Physicians Technical Equipment Company, Inc., New York, New York; Picker X-Ray Corporation, Cleveland, Ohio; Profexray, Inc., Maywood, Illinois; W. B. Saunders Company, Philadelphia, Pennsylvania; Schick X-Ray Company, Inc., Chicago, Illinois; Frank Scholz X-Ray Corporation, Boston, Massachusetts; Siemens Medical of America Inc., Union, New Jersey; Sigmamotor, Inc., Middleport, New York; Smith Kline—Precision Company, Philadelphia, Pennsylvania; E. R. Squibb & Sons, New York, New York; Standard X-Ray Company, Chicago, Illinois; Charles C Thomas, Publisher, Springfield, Illinois; U. S. Catheter & Instrument Corporation, Glens Falls, New York; United States Radium Corporation, Morristown, New Jersey; Westinghouse Electric Corporation, Baltimore, Maryland; The Williams & Wilkins Company, Baltimore, Maryland; Winthrop Laboratories, New York, New York; Year Book Medical Publishers, Inc., Chicago, Illinois.

NEWS ITEMS

EIGHTH INTERAMERICAN CONGRESS OF RADIOLOGY

The Eighth Interamerican Congress of Radiology took place at the luxurious Macuto Sheraton Hotel in Caracas, Venezuela, from October 2 to October 9, 1964, with Dr. César Herrera-Pinto as President and Dr. Rubén Merenfeld as Secretary-General, both of Caracas, Venezuela.

The Scientific Program, consisting of symposia, panel discussions and many papers on radiodiagnosis, radiotherapy and nuclear medicine was excellent; the Scientific and Technical Exhibits were beautifully displayed; and the social program surpassed all expectations in its warmth, splendor and Venezuelan hospitality. The Congress was very well attended and some important contributions were made from the United States.

The Interamerican College of Radiology held its sessions, as usual, during the Congress with Dr. Juan A. del Regato as Counselor for the United States. Dr. Oscar Soto of Lima, Perú, was elected the new President of the College. A Ceremonial Convocation of the College was held during the Congress and Honorary Fellowships were bestowed on Dr. César Herrera-Pinto of Caracas and Dr. Manuel Gaitan-Yanguas of Bogotá, Colombia. A Special Certificate of Appreciation was presented to Dr. M. Zariguiety.

At the closing ceremonies of the Congress, Honorary Fellowships in the Interamerican College of Radiology were bestowed on Dr. Manuel Viamonte, Sr., of Cuba, Dr. J. A. Saralegui, of Argentina, Dr. Ross Golden, of the United States, and Dr. Felix J. Daza, of Chile.

The Silver Medal of the College was awarded to Dr. W. Bomfim Pontes, of Brazil; and the Gold Medal to Dr. A. Frangella of Uruguay, Dr. J. M. Cabello Campos of Brazil, and Dr. Eugene P. Pendergrass of the United States.

The Ninth Interamerican Congress of Radiology will be held in Montevideo, Uruguay, in 1967.

AMERICAN ROENTGEN RAY SOCIETY ELECTS OFFICERS

At the Sixty-fifth Annual Meeting of the American Roentgen Ray Society held at the Radisson Hotel in Minneapolis, Minnesota, from Tuesday, September 29 through Friday, October 2, 1964, the following officers were elected: *President,* Dr. Harold O. Peterson, Minneapolis, Minnesota; *President-Elect,* Dr. J. Paul Medelman, White Bear Lake, Minnesota; *First Vice-President,* Dr. Carleton B. Peirce, Montreal, Quebec, Canada; *Second Vice-President,* Dr. Herman C. Sehested, Fort Worth, Texas; *Secretary,* Dr. C. Allen Good, Rochester, Minnesota; *Treasurer,* Dr. Stephen W. Brown, Augusta, Georgia.

Dr. J. Scott Dunbar, Montreal, Quebec, Canada, was elected to the Executive Council, filling the term of the deceased Dr. John D. Reeves, Gainesville, Florida.

Dr. Seymour F. Ochsner, New Orleans, Louisiana, was elected as the Representative of the American Roentgen Ray Society on the Board of Chancellors of the American College of Radiology.

The Sixty-sixth Annual Meeting of the Society will be held at the Hilton Hotel, Washington, D. C., September 28 to October 1, 1965.

SOCIETY FOR PEDIATRIC RADIOLOGY

At the Seventh Annual Meeting of the Society for Pediatric Radiology held on Monday, September 28, 1964, in conjunction with the Sixty-fifth Annual Meeting of the American Roentgen Ray Society, at the Radisson Hotel, Minneapolis, Minnesota, the following officers were elected: *President,* Dr. Edward B. Singleton, Houston, Texas; *President-Elect,* Dr. J. Scott Dunbar, Montreal, Quebec, Canada; *Secre-*

tary-Treasurer, Dr. John L. Gwinn, Children's Hospital, Los Angeles, California 90027.

The Eighth Annual Meeting of the Society will be held at the Hilton Hotel, Washington, D. C., September 27, 1965.

DYNAMIC FUNCTION STUDIES WITH RADIOISOTOPES

The Harvard Medical School Department of Radiology at the Peter Bent Brigham Hospital is sponsoring a symposium on "Dynamic Function Studies with Radioisotopes." This will be held at the Peter Bent Brigham Hospital on January 28, 29 and 30, 1965.

The subjects to be considered include: (1) diffusion and blood flow studies with radioactive cases; (2) isotopic studies of renal function and hypertension; (3) the gastrointestinal tract; and (4) isotopes in hematology.

There will be formal papers by an outstanding Faculty of Scientists from the United States, England and Denmark, followed by Round Table Discussions and Question Periods.

Inquiries for further information should be addressed to James B. Dealy, Jr., M.D., Radiologist-in Chief at the Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Massachusetts 02115.

QUEENS HOSPITAL CENTER

DEPARTMENT OF HOSPITALS THE CITY OF NEW YORK

A 4 month, 90 hour, course in the "Medical Uses of Radioactive Isotopes" will be offered at the Queens Hospital Center by the Radiation Medicine Department.

The course will last from February 2 through June 8, 1965, and will consist of weekly 5 hour sessions covering lectures, laboratory exercises and clinical management of patients.

The following topics will be covered: (1) diagnosis and therapy of thyroid disease; (2) isotope dilution techniques; (3) absorption studies; (4) organ scanning; (5) kidney and liver function; (6) blood dyscrasias; (7) circulation studies; (8) localization of tumors; (9) therapeutic uses of isotopes; (10) radiologic physics (instrumentation, dosimetry, health physics, laboratory design); (11) radiation biology; and (12) weekly laboratory workshop.

The enrollment is limited to 15.

For further information contact Dr. Leonard B. Goldman, Director, Radiation Medicine Department, Queens Hospital Center, 82-68 164th Street, Jamaica, N. Y. 11432.

UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL SCHOOL

The Department of Radiology of the University of Texas Southwestern Medical School, Dallas, Texas, will offer a four-year residency training program in radiotherapy, with initial appointments to be effective July 1, 1965. The new program will use the clinical and laboratory facilities of the following affiliated institutions: Baylor University Medical Center; Parkland Memorial Hospital; the U. S. Veterans Administration Hospital, all of Dallas; and the Radiation and Medical Research Foundation of the Southwest, Fort Worth.

Additional information about this residency may be obtained from John T. Mallams, M.D., Chairman of the Radiotherapy Training Committee, 3500 Gaston Avenue, Dallas, Texas, or Frederick J. Bonte, M.D., Chairman of the Department of Radiology, 5323 Harry Hines Boulevard, Dallas, Texas.

THIRD SYMPOSIUM CINERADIOGRAPHICUM

The Third Symposium Cineradiographicum will be held on May 28 and 29, 1965 in Antwerp, Belgium, on the premises of the Royal Society of Zoology, with Prof. Dr. S. Masy as President.

The languages of the Symposium will be English, French and German. The method adopted in Munich, to project on two large screens the summary of the paper in the two languages not used by the author, will be continued. It is therefore requested that the summary of the paper be submitted not later than March 31, 1965, with an indication whether 10 or 20 minutes are desired for the paper's presentation.

Cineradiographic films already shown at other International Congresses will not be accepted.

Authors who wish to present papers or cineradiographic films are invited to submit the title and an abstract of the subject before January 1, 1965.

For further information please write to Dr. Sc. Ph. Lauwers, General Secretary of the Third Symposium Cineradiographicum, L. Gerritslaan 35, Berchem-Antwerpen, Belgium.

COURSE IN PEDIATRIC RADIOLOGY, INDIANA UNIVERSITY

The Department of Radiology of Indiana University School of Medicine announces a postgraduate course April 6–10, 1965. The course is planned primarily for the general radiologist but is open to all physicians having an interest in this field. Presentations will consist of lectures, film interpretation conferences, and exhibits correlating the radiologic, pathologic, and physiologic aspects of various pediatric conditions encountered in clinical practice.

It will be presented by the faculty of Indiana University and the following guest participants: David H. Baker, New York City; John Caffey, Pittsburgh; Robert Evans, Houston; Bertram Girdany, Pittsburgh; John L. Gwinn, Los Angeles; H. J. Kaufmann, Basel, Switzerland; John E. Moseley, New York City; Charles E. Shopfner, Kansas City, Mo.; Frederick N. Silverman, Cincinnati; Edward B. Singleton, Houston; Hooshang Taybi, San Francisco; and M. H. Wittenborg, Boston.

For further information, write the Director of Postgraduate Medical Education, Indiana University School of Medicine, Indianapolis, Indiana, 46207.

THE ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

At the recent Annual Meeting of the Rocky Mountain Radiological Society, the following officers were elected for 1965: *President*, Morris H. Levine, M.D., Denver, Colorado; *President-Elect*, Lester D. Shook, M.D., Fargo, North Dakota; *1st Vice-President*, Robert W. Lackey, M.D., Denver, Colorado; *2nd Vice-President*, John R. Danstrom, M.D., Oklahoma City, Oklahoma; *Secretary-Treasurer*, John H. Freed, M.D., Denver, Colorado; and *Historian*, H. Milton Berg, M.D., Bismarck, North Dakota.

The next annual meeting of the Society will be held in Denver at the Brown Palace Hotel on August 19, 20 and 21, 1965.

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BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

RADIOLOGIA INFORTUNISTICA DELLA COLONNA VERTEBRALE: PATOLOGIA TRAUMATICA E MEDICINA LEGALE. By Lorenzo Giuntoli, and Bruno Guareschi, Instituto di Radiologia dell'Università di Milano, Italy. Cloth. Pp. 436, with 272 illustrations. Price, Lire, 12.000. L. Cappelli, Via Farini 6, Bologna, Italy, 1963.

This is an important book written by two authors of obvious competence and vast experience in traumatic radiology. Both are connected with the Medical School of the University of Milan and work with The Italian Workmen's Compensation System. This excellent treatise is difficult to abstract because the authors consider in great detail and illustrate with beautiful roentgenograms and schematic drawings practically all the fractures, dislocations and soft tissue injuries which occur in the spinal structures. They also describe the healing sequence of each injury and give useful signs for the roentgenologic determination of the approximate time of the trauma.

The printing is modern, and the illustrations are superb. The text is clear and concise.

The authors begin by describing the embryology of the spine and its later development, including not only the bony structures but also the articular tissues and the neural components. Chapters are devoted to injuries of the vertebral bodies in each of the spinal segments, to injuries of the neural arches and articular facets, and to injuries of the intervertebral disks. Especially good are those chapters in which the injuries of the cervical area and of the sacrococcygeal segments are described. The differential diagnosis of injuries versus malformations is thoroughly discussed as well as the effect of injuries on malformed spines. Spondylolisthesis in its anterior, lateral and posterior varieties is presented with numerous roentgenograms, drawings and laminagrams and its medico-legal significance is fully evaluated.

In several chapters on differential diagnosis, osteoporosis, inflammatory and neoplastic diseases are considered. The congenital and hereditary nature of some malformations is again discussed in connection with the studies of Kuhne on the search of paternity by radiologic means.

This volume is a veritable mine of information for those radiologists who deal with accident and compensation cases. It is to be hoped that the authors will find it possible to publish their book in the English language in the near future.

CESARE GIANTURCO, M.D.

BOOKS RECEIVED

PROGRESS IN ANGIOGRAPHY. Compiled and Edited by Manuel Viamonte, Jr., M.D., Associated Professor of Radiology, University of Miami School of Medicine, Miami, Fla.; and Raymond E. Parks, M.D., Professor and Chairman of Radiology, University of Miami School of Medicine, Miami, Fla. Cloth. Pp. 562, with many illustrations. Price, $29.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1964.

RADIOISOTOPES AND RADIATION: RECENT ADVANCES IN MEDICINE, AGRICULTURE, AND INDUSTRY. By John H. Lawrence, M.D., Director, Donner Laboratory and Donner Pavilion, University of California, Berkeley, Calif.; Bernard Manowitz, Head, Radiation Division, Nuclear Engineering Department, Brookhaven National Laboratory; and Benjamin S. Leob, Assistant to the Director, Division of Technical Information, United States Atomic Energy Commission. Cloth. Pp. 131, with many illustrations. Price, $18.00. McGraw-Hill Book Company, 330 West 42nd Street, New York 10036, 1964.

SURGICAL DISEASES OF THE LIVER. By Seymour I. Schwartz, M.D., F.A.C.S., Associate Professor of Surgery, Director of Surgical Research, John and Mary R. Markle Scholar in Academic Medicine, University of Rochester School of Medicine and Dentistry; Senior Associate Surgeon, Strong Memorial Hospital, Rochester, N.Y. Cloth. Pp. 387, with many illustrations. Price, $17.50. McGraw-Hill Book Company, 330 West 42nd Street, New York, 10036, 1964.

SCHEURERMANN'S JUVENILE KYPHOSIS: CLINICAL APPEARANCES, RADIOGRAPHY, AETIOLOGY, AND PROGNOSIS. By K. Harry Sørensen. Paper. Pp. 273, with some illustrations. Price, $6.00. Munksgaard, 47, Prags Boulevard, Copenhagen, Denmark, 1964.

Handbuch der Tuberkulose. In fünf Bänden. Edited by Prof. Dr. J. Hein, Direktor des Krankenhauses Tönscheide/Holstein; Prof. Dr. Dr. h.c. H. Kleinschmidt, ehemals Direktor der Kinderklinik der Universität Göttingen, Bad Honnef; and Prof. Dr. E. Uehlinger, Direktor des Pathologischen Institutes der Universität Zürich. Band IV: Formenkreis der extrapulmonalen hämatogenen Tuberkulose. Cloth. Pp. 955, with 438 illustrations and 68 tables. Price, DM 248.-. Georg Thieme Verlag, 7000 Stuttgart 1, Postfach 732, Herdweg 63, Germany, 1964.

Staublungenerkrankungen und ihre Differentialdiagnose. Edited by Dr. med. H. Bohlig, Chefarzt der Strahlenabteilung, Städtisches Krankenhaus Lüdenscheid/Westf. Cloth. Pp. 386, with 196 illustrations. Price, DM 150.-. Georg Thieme Verlag, 7000 Stuttgart 1, Postfach 732, Herdweg 63, Germany, 1964.

Shielding for High-Energy Electron Accelerator Installations. National Bureau of Standards Handbook 97. Paper. Pp. 65, with many figures and tables. Price $0.30. For sale by Superintendent of Documents, U. S. Government Printing Office, Washington, D. C., 20402, 1964.

Dynamic Clinical Studies with Radioisotopes. Proceedings of a symposium held at the Oak Ridge Institute of Nuclear Studies, Oct. 21–25, 1963. Edited by Ralph M. Kniseley, M.D., and W. Newlon Tauxe, M.D.; Technical Editor, Elizabeth B. Anderson, M. S. Paper. Pp. 632, with many figures and tables. Price, $4.50. Available from Office of Technical Services, U. S. Department of Commerce, Washington, D.C., 20230, 1964.

La Scintigraphie du Corps Thyroïde. By J. C. Savoie, Service Central des Radio Isotopes du Groupe Hospitalier, La Pitié—La Salpétrière, Paris; and G. Vallee, Service Central des Radio Isotopes du Groupe Hospitalier, Necker—Enfants Malades, Paris. Collection Médicale Internationale "Savoir Interpreter," No. 28. Paper. Pp. 112, with 84 figures. Price, 120 FB. Albert de Visscher, Editeur, 65, Avenue du Golf, Rhode-St.-Genèse, Belgium, 1964.

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ABSTRACTS OF RADIOLOGICAL LITERATURE

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

HEAD

STENGER, J. M., *et al.:* Mouthguards: protection against shock to head, neck and teeth 1432
PINET, F., and PINET, A.: Dental lesions in chronic fluorosis; origin of the Sahara fluorine and its aggressiveness 1432
SUBIRANA, M., and VROUSOS, C.: Frontal pneumostratigraphy of the cerebellar hemispheres 1432
PORTERA, A.: Panventriculography: a new method with emulsified pantopaque 1433
CORTES, O., CHASE, N. E., and LEEDS, N.: Visualization of tentorial branches of the internal carotid artery in intracranial lesions other than meningiomas 1433
WHEELER, E. C., and BAKER, H. L., JR.: The ophthalmic arterial complex in angiographic diagnosis 1433
ARDRAN, G. M., BECKETT, J. M., and KEMP, F. H.: Aglossia congenita 1434

NECK AND CHEST

KANDT, D., and SCHOEFER, G.: Rust granuloma of the lung: contribution to the corroding process of metallic splinters in the body . . 1434
LENCZNER, M., SPAULDING, W. B., and SANDERS, D. E.: Pulmonary manifestations of parasitic infestations 1435
MARSHALL, W. C., WESTON, H. J., and BODIAN, M.: Pneumocystis Carinii pneumonia and congenital hypogammaglobulinaemia 1435
MORI, P. A., ANDERSON, A. E., JR., and ECKERT, P.: The radiological spectrum of aging and emphysematous lungs 1436
ROSENBAUM, H. T., THOMPSON, W. L., and FULLER, R. H.: Radiographic pulmonary changes in near-drowning 1436
NATHAN, M. H.: The diagnosis of dehydration, acidosis, and gastroenteritis in infants from the chest radiograph 1436
CHOREMIS, C., *et al.:* Spontaneous elimination of inhaled grass inflorescences through the lung and chest wall 1437
SCHNEIDER, L., and WIMPFHEIMER, F.: Multiple progressive calcific pleural plaque formation: a sign of silicatosis 1437
KREEL, L., BLENDIS, L. M., and PIERCY, J. E.: Pneumo-mediastinography by the trans-sternal method 1437

ABDOMEN

DELAHAYE, R. P., *et al.:* Chronic intestinal amebiasis: radiologic study of 171 cases 1438
BRUNTON, F. J.: Choristoma of the gallbladder 1438
HAASTERT, S.: Tolerability and diagnostic results of the clinical trial of bilivistan (SH 419) 1439
ELKINGTON, S. G., and BERNSTEIN, D. C.: Percutaneous transhepatic cholangiography 1439

GENITOURINARY SYSTEM

CRAVEN, J. D.: Renal glomerular osteodystrophy 1440
SCHENCKER, B.: Drip infusion pyelography: indications and applications in urologic roentgen diagnosis 1440
BLISS, B. P., CLARK, W. G. B., and SAUNDERS, M.: Recurring spontaneous rupture of the urinary bladder following subarachnoid haemorrhages 1440

Vermooten, V., and Schweinsberg, M.: Radiographic estimation of the size of the prostate 1441

Skeletal System

Ferrand, J., *et al.:* Albuminous periostitis of Ollier and Poncet (segmental angiographic studies and abcessography) 1441
Carbonara, P., and Alpert, M.: Hereditary osteo-onycho-dysplasia (HOOD) 1441

RADIOISOTOPES

Entzian, W., *et al.:* A preliminary evaluation of F^{18}-labeled tetrafluoroborate as a scanning agent for intracranial tumors 1442

ROENTGEN DIAGNOSIS

HEAD

STENGER, JOHN M., LAWSON, EDWARD A., WRIGHT, JACK M., and RICKETTS, JAMES. Mouthguards: protection against shock to head, neck and teeth. *J. Am. Dent. A.*, Sept., 1964, *69*, 273–281. (Address, 2217 Lincoln Way West, South Bend, Ind.)

The repositioning of the mandibular condyle, cervical vertebrae and other anatomic structures, brought about by acrylic template and mouthguard, is shown graphically in tracings of the cephalometric roentgenograms of football players.

The plan was to assess, initially, the general extent of use of mouthguards by players and their attitudes toward them; to review the 3 types of mouthguards available—stock, mouth-formed, custom-made—to ascertain the most effective type, and then to determine in practical tests what effect if any it had in preventing and reducing injuries.

A mouthguard must: (1) be custom-made to an accurate model of the player's mouth; (2) be comfortable in wearing so that the player will accept it; edges should be thin; (3) have sufficient retention to prevent accidental dislodgment during athletic contact or calling signals; (4) be tough enough to prevent cuspal penetration; (5) be thermally resistant to enable sterilization by boiling or autoclaving and, also, be compatible with the oral tissue at cool temperatures; (6) be chemically odorless and tasteless; and (7) receive approval of all components, singly and in combination, by the Food and Drug Administration.

During 1963 spring practice, however, an entirely new type of mouthguard was designed. It was a custom-made modified vinyl elastomer guard and proved extremely successful. As expected, injuries to the teeth and jaws were nil. Even more important, however, was the impressive reduction in concussions.

The success of this mouthguard in reducing injuries led to its use by the Notre Dame team during the entire 1963 football season.

The following is a summary of the teeth, head and neck injuries reported:

Injury to the Dentition. All 4 minor teeth injuries that occurred during the 1963 season involved boys who were not wearing mouthguards.

Head Injury. There were 10 concussions, and in only 1 instance with the player wearing a mouthguard.

Neck Injury. A reduction in the number of neck injuries was an unexpected result of wearing the mouthguards. Not a single Notre Dame player who faithfully wore his mouthguard during the 1963 season found it necessary to wear a cervical collar.

Case reports, with the roentgenographic reproductions and tracings, illustrate the method of study which produced the results described.

By using this mouthguard, the patients were all relatively free of any type of disability from such injuries.—*Stephen N. Tager, M.D.*

PINET, F., and PINET, A. (Lyon, France). Lésions dentaires des fluoroses chroniques: origine du fluor au Sahara et son aggresivité. (Dental lesions in chronic fluorosis; origin of the Sahara fluorine and its aggressiveness.) *Presse méd.*, April 22, 1964, *72*, 1205–1210.

The authors allude to the controversial nature of the subject. This report is based on a study of 3,000 people living in 7 oases of Sahara Desert; 2,520 of them were students ranging from 6 to 16 years of age. The concentration of fluorine varied from 0.3 mg. to 4 mg. per liter of water from one oasis to another. Thus, a controlled experimental gradation of fluorine content was present for a comparative study.

The deciduous teeth were unaffected. Dental caries was noted in 20 per cent of people living in a region where the fluorine concentration of water was below 0.5 mg. per liter, 95 per cent of caries where water showed 0.8 mg. per liter and 100 per cent where concentration reached 1 mg. per liter of water.

The permanent lesions were those of the enamel, whereas gingivitis and periodontal involvements were transient and noted only in cases of serious fluorine intoxication. The enamel lesions could be noted as white or as brown spots. These fluorine spots resulted only where the subject consumed water containing a high percentage of fluorine before the eruption of the permanent teeth.

Many people who came to live in regions with high fluorine content of water after the age of 10 or 12 did not develop any enamel lesion even though the concentration of fluorine in the teeth themselves rose to a high level. This points the avidity of dental tissue for this metalloid.

Other factors are poor protein intake and individual variation in susceptibility; hence, the difficulty of arriving at an optimal dose of water fluorine content for tooth care. The border line between the optimal beneficial dose and toxic concentration is so close that the authors warn of the risks, especially since many aspects of fluorine metabolism are unknown.—*Jirair N. Sarian M.D.*

SUBIRANA, M., and VROUSOS, C. Pneumostratigraphie frontale des hémisphères cérébelleux. (Frontal pneumostratigraphy of the cerebellar hemispheres.) *J. de radiol., d'électrol. et de méd. nucléaire*, May, 1964, *45*, 225–232. (From: Laboratoire de Radiologie de la Clinique Neurologique de Strasbourg, France.)

The authors routinely use at the start of all air encephalographic studies, the easy technique of median sagittal tomography, previously described by Thiébaut, Wackenheim and Vrousos in this same

Journal in 1959. This technique results in useful information on the cerebello-bulbo-cervical region, the ventricular channel, the vermis, the prebulbar and prepontine cisterns. The paramedian sections do not furnish valuable information on the cerebellar hemispheres.

This article presents an investigation on frontal tomography of the cerebellar fossae. The technique consists of a second injection of 10 cc. of air with the patient's head overflexed, immediately following the initial phase in the sitting position, and after control with median sagittal tomography. Then frontal tomography is performed in the plane of the external auditory meati. This is followed by 4 to 6 sections more posteriorly, ½ cm. apart. These tomograms show the air around the cerebellar formations. The ideal projection of this pericerebellar air will be between the lateral ventricles above and the facial bones below. To obtain this result, a horizontal beam is used, with the head flexed so that Reid's line makes a 25° angle with the horizontal.

The authors demonstrate with roentgenographic reproductions and schematic drawings the normal anatomy of these different tomographic sections. They present a case of cerebellar atrophy.—*H. P. Lévesque, M.D.*

Portera, Alberto. Pan-ventriculografía: nuevo método con pantopaque emulsionado. (Panventriculography: a new method with emulsified pantopaque.) *Acta iber. radiol. cancerol.*, July–Sept., 1964, *19*, 221–229.

This new method of ventriculography consists of injecting into the ventricular system an emulsion of pantopaque with cerebrospinal fluid (CSF).

The technique is as follows: Under local anesthesia a burr hole is drilled, usually, in the right frontal region. A catheter is passed into the frontal horn of the ventricular system, and 8 to 15 ml. of CSF is aspirated. While the catheter is occluded to prevent escape of more CSF, 1.5 to 3 ml. of pantopaque is introduced into the syringe, as well as some 5 ml. of air. The mixture is vigorously shaken to produce a milky emulsion. The air is then expelled, the syringe is reconnected to the catheter, and the emulsion rapidly injected. Two seconds later a lateral skull roentgenogram is taken, and afterwards another one in an anteroposterior projection. The constrast medium fills the entire ventricular system, including the Monro foramina, third ventricle with all its recesses, sylvian aqueduct, fourth ventricle, cisterna magna, and upper cervical region.

This technique has been carried out on 43 patients with excellent results. The entire ventricular system can be visualized on a single roentgenogram, without having to move the patient's head. The ventriculograms show very good detail, which makes for easy interpretation. No cases of severe headache or shock have been observed, due to decompression, because no CSF need to be removed and only 1 to 3 ml. of contrast medium is injected. The procedure is painless. All patients showed a slight febrile reaction (37.5 to 38.5° C.) for the next 12 to 24 hours; in 1 child the temperature reached 39.5° lasting for 48 hours. No other complications have occurred.

The roentgenographic reproductions show beautiful filling of the ventricular system.—*Frank V. Comas, M.D.*

Cortes, Octavio, Chase, Norman E., and Leeds, Norman. Visualization of tentorial branches of the internal carotid artery in intracranial lesions other than meningiomas. *Radiology*, June, 1964, *82*, 1024–1028. (From: Department of Radiology, New York University, New York 16, N. Y.)

The purpose of this paper is amply stated in the title. The authors have observed the tentorial branch or branches of the internal carotid artery in 3 arteriovenous malformations and 1 malignant glioma. From their experience they consider it of value in localizing an intracranial lesion adjacent to the tentorium but useless in establishing a histologic diagnosis.

In 1956, Bernasconi and Cassinari demonstrated a tentorial artery in 5 of 7 tentorial meningiomas and postulated its specificity in the angiographic diagnosis of these tumors. This postulation was somewhat repudiated in 1957 by Krayenbuhl and Yasargil and again in 1961 by Stattin who demonstrated the artery in several cases of arteriovenous malformation. In 1963, Schnurer and Stattin injected the internal carotid artery at the base of the skull in autopsy specimens and described in detail the anatomy of the tentorial artery. Usually, 2 main branches were found.

The tentorial artery is rarely visualized in the normal subject and then will be thread-like, but will have a fairly wide caliber in pathologic conditions. It arises from the intracavernous portion of the internal carotid artery and courses convexly posteriorly and slightly laterally, though it maintains a relatively medial position. To prove its exact source from the internal carotid artery, either pure injection of the internal carotid artery must be done in order to avoid superimposition of external carotid artery branches, or, serial films are required. The authors employed simultaneous biplane rapid serial angiography. Films were programmed at 2 per second for the initial 3 seconds and 1 per second for 4 more seconds. A Taveras injector at 225 pounds pressure delivered 8 ml. of contrast material in 1½ seconds.—*Howard R. Stewart, M.D.*

Wheeler, Edward C., and Baker, Hillier L., Jr. The ophthalmic arterial complex in angiographic diagnosis. *Radiology*, July, 1964, *83*, 26–35. (From: Mayo Foundation and Mayo Clinic, Rochester, Minn.)

The clinical course of all intraorbital space occupying lesions is essentially the same with progressive unilateral proptosis, disturbance of ocular motility and perhaps decreased vision and mild pain. If physical examination and plain skull or orbital roentgenograms fail to establish the presence of the lesion, visualization of the ophthalmic arterial complex on carotid angiography can be of immense value.

To clarify the normal angiographic features of this arterial group, 300 angiograms were reviewed. An excellent analysis and description of the angiographic anatomy is presented, along with the normal variations.

The authors observed that the most effective opacification of the orbital vessels was produced by injection of larger volumes(10–12 ml.) of contrast material into the carotid artery, and additional exposures in the 2 to 5 second interval after injection.

In order to evaluate the abnormal changes, 57 lesions involving the orbit or ophthalmic arterial complex were reviewed. These consisted of a diverse group of both primary and secondary tumors of the orbit along with vascular tumors and anomalies. The angiographic findings were positive in 44 lesions and one or more of the following abnormalities were noted: (a) displacement of the arterial system, (b) intrinsic alterations in the arteries characterized by dilatation (aneurysmal, beaded or diffuse), and (c) pathologic vascularity of the lesion which also gave a clue as to its benign or malignant nature. These changes are excellently described and illustrated and study of the original article is recommended.—*E. B. Best, M.D.*

ARDRAN, G. M., BECKETT, J. M., and KEMP, F. H. Aglossia congenita. *Arch. Dis. Childhood*, Aug., 1964, *39*, 389–392. (From: Nuffield Institute for Medical Research, University of Oxford, and the Health Department, Nuneaton, England.)

The authors describe their observations in a child of 8 years and 6 months of age with aglossia congenita, whom they have followed since birth. The child was first observed to have no tongue at all as far as the fauces, and only on the lateral roentgenogram was a posterior segment demonstrated. At the age of 8 years, the tongue has tended to grow forward and to have sufficient mobility together with the very mobile floor of the mouth to make substitute movements that mimic a normal tongue fairly closely.

For speech purposes, those actions usually performed with the tip of the tongue take place between the tongue and the hard palate and are made further back than is customary. It appears, therefore, that a child with this disability can be given a good prognosis as to swallowing and speech, provided that his initial difficulties with sucking and feeding in the early weeks can be understood and overcome. The relatively normal speech has been achieved without speech therapy.

Cineroentgenographic observations are presented with the child breathing, phonating and swallowing. —*James F. Martin, M.D.*

NECK AND CHEST

KANDT, D., and SCHOEFER, G. Das Rostgranulom der Lunge: Ein Beitrag zum Verrottungsvorgang von Metallsplittern im Körper. (Rust granuloma of the lung: contribution to the corroding process of metallic splinters in the body.) *Thoraxchirurgie*, Aug., 1964, *12*, 81–86. (Address: Dr. med. G. Schoefer, Heilstätte Vogelsang-Gommern bei Magdeburg, Germany.)

Metallic foreign bodies may remain asymptomatic in the lungs for a long time, but when recurrent hemorrhages, pulmonary abscess and gangrene as well as pleural complications with empyema develop, surgical measures become necessary. Other indications for operations are increased size of the splinter, also bronchial changes like bronchostenosis and bronchiectasis.

Preoperatively, precise localization of the foreign body and of the perifocal inflammation is essential. Laminagrams in two planes and bronchoscopic as well as bronchographic studies are most helpful. Two pertinent cases are reported.

The first patient had a grenade splinter in the right upper lobe for 22 years. For the past 6 years, he complained of cough and hemoptysis which required repeated hospitalization. Within a period of 3 years, the splinter and the perifocal infection increased in size. The involved lobe was resected, and the specimen displayed a cherry-sized metallic foreign body with a rusted shell and perifocal infection of tissues containing iron pigment.

The second patient also had a grenade splinter in his left upper lobe for 17 years, which gave rise to occasional coughing spells and to a pneumonic process 4 years ago. More recently, a hemoptysis occurred. Serial roentgenograms made since 1954 disclosed a growing small metallic foreign body in the left upper lobe surrounded by an ever increasing zone of pneumonitis. Laminagraphic studies suggested that the splinter was lodged in a cavity. A lobectomy was performed, disclosing a corroded metallic foreign body in a small cavity. The surrounding infected lung tissues contained iron pigment which suggested decomposition of the foreign body.

It is postulated that the high oxygen content of the lung tissues gives rise to an electrochemical reaction, thereby causing decomposition of the iron.—*Ernest Kraft, M.D.*

LENCZNER, M., SPAULDING, W. B., and SANDERS, D. E. Pulmonary manifestations of parasitic infestations. *Canad. M. A. J.*, Aug. 29, 1964, *91*, 421–434. (From: Medical Out-Patient Department, Toronto General Hospital, Toronto, Ontario, Canada.)

Parasitic infestations, which may present as transient or permanent pathologic changes in the lungs, often pose difficult diagnostic problems. These parasitic infestations have no distinctive clinical traits; therefore, the search for the parasite is often delayed until exhaustive diagnostic procedures have failed to identify a more common and familiar condition. Parasites may reach the lungs during migration and evolution in the human body, during a parasitemia, or as a final habitat. Pulmonary invasion may be accompanied by irritating cough especially at night, dyspnea, asthma, blood-stained expectoration and pain in the chest. The symptoms may be transitory or prolonged, with or without systemic disturbances which may be serious, and associated with sensitivity reactions such as urticaria and eosinophilia.

The parasitic infestations causing pulmonary manifestations are: (1) ascariasis; (2) strongyloidiasis; (3) ancylostomiasis (*Ancylostoma duodenale*, *Necator americanus*); (4) filariasis (*Filaria* [or *Wuchereria*] *bancrofti*, *malayi* or *Loa loa*); (5) pulmonary distomiasis; and (6) hydatid disease.

The transient as well as permanent pulmonary manifestations are described in detail on the basis of case reports.

1. *Pneumonic disease*. Loeffler's pneumonia may occur with many parasitic infestations and is not uncommonly encountered when roundworm larvae migrate through the lungs.

Nonspecific pneumonic infiltrates are the rule, but occasionally severe pulmonary edema and interstitial pulmonary hemorrhage may occur (ascariasis, strongyloidiasis). Pleural reactions (ancylostomiasis, hydatid disease, amebiasis) and effusions may also occur.

2. *Disseminated ("miliary") lesions*. Infestations with schistosomiasis will on occasion produce a diffuse miliary infiltrate, which is less common in other parasitic diseases. This type of reaction may provoke a chronic interstitial pneumonitis which heals by fibrosis.

3. *Bronchomediastinal lymphadenopathy*. Enlargement of the hilar lymph nodes often produces a readily recognized roentgen picture, but this, too, is a relatively nonspecific finding. Of the parasitic diseases, filariasis may produce roentgenologically evident enlargement of the bronchial and/or mediastinal lymph nodes.

4. *Parenchymal and mediastinal cysts*. When hydatid cysts occur in the lungs, mediastinum or heart, the roentgenologic diagnosis may be highly accurate. These cysts are round or oval in shape, are sharply defined, and may show a distinct alteration in shape with forced inspiration and expiration. They may be single or multiple, may rupture, evacuate or become secondarily infected. The cyst wall, which rarely calcifies in the chest, is made up of two layers. The outside fibrous layer is formed by host tissues, the inner by the parasitic membrane. Collections of air may separate the two layers to produce a halo or crescentic effect, described by Kerley as pericystic emphysema. Another helpful sign in diagnosis is the tendency of cysts, when multiple, to adhere together and when touching, to have flattened contacting surfaces. Cysts can cause displacement of adjacent structures or erosion of ribs, sternum, etc. More rarely an alveolar type of hydatid disease may occur which does not exhibit typical signs on the roentgenogram.

Other pulmonary manifestations may be observed in:

Amebiasis. In the severe complications of this disease, rupture of amebic liver abscesses into the pleural space and lung may occur; secondary lung abscess, pleural effusion, empyema and bronchohepatic fistulas may result.

Paragonimiasis (lung fluke). This disease is characterized by a peripherally located, small, round or oval cyst measuring 1 to 2 cm. in diameter. The cyst wall is thin above (1–2 mm.) and thicker below (3–4 mm.) and may be surrounded by a narrow ring of pericystic inflammatory reaction. Pleural thickening is frequent.

Soft tissue calcification may occur in trichinosis in intercostal and diaphragmatic muscles, but the intrathoracic structures are not involved. In cysticercosis, calcified larvae may occur in the paraspinal muscles and around the shoulder girdle.—*Stephen M. Tager, M.D.*

MARSHALL, W. C., WESTON, H. J., and BODIAN, MARTIN. Pneumocystis Carinii pneumonia and congenital hypogammaglobulinaemia. *Arch. Dis. Childhood*, Feb., 1964, *39*, 18–25. (From: The Hospital for Sick Children, Great Ormond Street, London, England.)

Two case histories of patients with congenital hypogammaglobulinemia who developed Pneumocystis Carinii pneumonia are described. Both occurred in males, one at the age of 6 months and the other at the age of 2 years. The clinical features included an insidious onset with troublesome cough and respiratory distress progressing to extreme tachypnea and cyanosis. The temperature was either normal or slightly raised throughout the illness. A high leukocytosis was present with a preponderance of polymorphous neutrophils.

The chest roentgenograms in the 2 cases demonstrated a fairly distinctive pattern with widespread infiltration of the lung fields, most striking in the hilar region. The process was diffuse and not lobar in distribution and there was no involvement of the pleura. An "air bronchogram" was visible.

The occurrence of cases during an epidemic with the appearance of the typical clinical picture and roentgenographic changes makes for easy clinical diagnosis. Isolated cases must be differentiated from lipoid pneumonia, primary atypical pneumonia, Friedländer's pneumonia and such conditions as pulmonary hemosiderosis, diffuse pulmonary fibrosis of the Hamman-Rich type and histiocytic reticuloendotheliosis.

Diagnosis of this condition seems to be only satisfactorily made by lung biopsy.

In one case, paper electrophoresis of the serum revealed an apparently normal gamma globulin band in spite of the fact that immunochemical methods gave very low values for the gamma globulin; this was at least partly due to very high concentration of gamma macroglobulin.

A great variety of agents have been used in the treatment of this condition and many have not been successful. The authors report one case which was treated with pentamidine and gamma globulin which appeared to be effective.—*James F. Martin, M.D.*

MORI, PAUL A., ANDERSON, A. E., JR., and ECKERT, PHILLIPPA. The radiological spectrum of aging and emphysematous lungs. *Radiology*, July, 1964, *83*, 48–57. (From: Departments of Radiology and Research, Baptist Memorial Hospital, Jacksonville, Fla.)

A study was performed to ascertain the roentgenologic features of emphysematous and normal aging subjects on plain films, the ability to detect such changes and the reliability of present criteria in differentiating aging and emphysematous patients. Eleven radiologists participated in the study. There were 26 normal and emphysematous subjects ranging in age from 30 to 71 years. Posteroanterior and lateral chest roentgenograms in inspiration and expiration were obtained in each case.

Traditionally, radiologists have diagnosed emphysema on the basis of various combinations of the following signs: increased anteroposterior diameter of chest; increased retrosternal space; low position of diaphragm; narrow heart, increased prominence of pulmonary artery and major branches; decreased vascularity of peripheral lung fields; diminished range of excursion of diaphragm; bullous spaces in the lungs.

Hyperinflation of the lungs, increased translucence of lung fields, and decreased excursion of the diaphragm were features of both normal aging and pulmonary emphysema. Bullous spaces were found in the lungs of normal and emphysematous patients, but their size was not reflected in pulmonary function tests. Prominent pulmonary arteries were observed in every patient with severe emphysema but were also seen in some normal controls and in some patients with mild and moderate emphysema. Severe generalized emphysema sometimes occurred without roentgenographic evidence of pulmonary hyperinflation. Moderate and severe pulmonary emphysema was readily recognized in the presence of pulmonary hyperinflation. Serrations of the diaphragm were found only in patients with moderate or severe emphysema.

The study indicates that criteria generally used by radiologists, although they serve reasonably well in identifying moderate and severe emphysema, are unreliable in differentiating normal aging patients from those with mild emphysema.—*A. W. Sommer, M.D.*

ROSENBAUM, HOWARD T., THOMPSON, WILLIAM L., and FULLER, ROGER H. Radiographic pulmonary changes in near-drowning. *Radiology*, Aug., 1964, *83*, 306–313. (From: Armed Forces Institute of Pathology, Washington, D. C. 20305.)

The roentgenographic features of 10 cases of near-drowning, 1 case of sea-water aspiration, and 2 of acute death from drowning are presented. This series tends to support previous opinion that the most frequent roentgenographic appearance is that of a diffuse, fuzzy, nodular infiltration throughout both lung fields. Changes are more concentrated in the perihilar and medial basilar areas and are less marked in the apices, the extreme lateral portions of the lungs and bases. Less frequently there may be a fairly homogeneous, diffuse infiltration with small, fuzzy nodules covering almost the entire lung field but with minimal involvement of the extreme bases and apices. The process is bilateral but may be more marked in one lung. The infiltration tends to clear almost entirely in 12 hours to 6 days in the uncomplicated case. No difference was noted in the roentgenographic findings in patients submerged in fresh water and those who aspirated sea water.

One patient, a 21 year old white male who suffered near-drowning in swamp water, was treated with penicillin and streptomycin but continued to be febrile for the first 4 days. A roentgenogram 26 days postsubmersion showed some residual, bilateral, lower lung field streaking.

Judging from the roentgenographic appearance of these near-drowning cases and the roentgen appearance and autopsy findings in the lungs in the 2 cases of acute death from drowning, the authors conclude that the roentgenographic findings represent pulmonary edema and hemorrhage.—*A. W. Sommer, M.D.*

NATHAN, M. H. The diagnosis of dehydration, acidosis, and gastroenteritis in infants from the chest radiograph. *Radiology*, Aug., 1964, *83*, 297–305. (From: Baptist Hospital of Scottsdale, Scottsdale, Ariz.)

The clinical complex of gastroenteritis, dehydration and acidosis in infants and young children may often be accurately suggested by the chest roentgenogram alone.

Hyperventilation is the most consistent and reliable roentgenographic sign of dehydration and acidosis. Its earliest recognition depends on elevation and anterior bowing of the sternum. Dehydration is expressed roentgenographically by the loss of soft-tissue thickness. The thinning of the soft tissues of the lateral chest wall is easily recognized and is a reliable sign of dehydration. The most common roentgenographic sign of enteritis is distention of the bowel by air and fluid. The roentgen appearance may suggest partial obstruction or paralytic ileus. Diminished bowel gas is less commonly seen.

The roentgenographic syndrome of hyperventilation, thinning of the soft tissues, and bowel signs of enteritis permits the diagnosis of dehydration and acidosis to be made with considerable accuracy in the absence of any clinical information.

Five cases are reported to illustrate the roentgenographic syndrome of hyperventilation, dehydration and diarrhea.—*A. W. Sommer, M.D.*

CHOREMIS, C., THEODOROU, S., ATHANASIADES, TH., and KATERELOS, H. Spontaneous elimination of inhaled grass inflorescences through the lung and chest wall. *Arch. Dis. Childhood*, Aug., 1964, *39*, 406–408. (From: Paediatric Clinic of University of Athens, Athens, Greece.)

The fate of inflorescences entering the respiratory tract depends upon their botanic characteristics which produce two distinct clinical types. In the first, because of their construction, inflorescences remain in the bronchi until removed bronchoscopically; in the second, to which the majority of cases belong, inflorescences, owing to their ability to travel towards the periphery, penetrate the parenchyma of the lung, the pleural layers and the intercostal muscles and finally protrude under the skin of the chest wall.

The authors report 2 cases of inhaled grass inflorescences that were spontaneously eliminated from the thoracic wall: one in a 3 year old girl, and the second in a 2½ year old boy. In both cases, the inhaled grass inflorescences migrated through the bronchi, lung, and chest wall and reached the skin. The migration was completed in about 2 months in the first patient, but took only 10 days in the second. The longer sojourn of the foreign body in the respiratory tract in Case I was due to the fact that the wheat stem, deprived of spikelets, was driven slowly towards the periphery, while the arrangement of the spikelets and the elasticity of the stem in Case II gave to the inflorescence a particularly fast propulsive ability. Both grasses are extremely common in Greece. In fact, children amuse themselves by demonstrating the crawling qualities of inflorescences like barley, wheat, and particularly Hordium murinum which was found in Case II, and which grows in almost every field in Greece. Children place the spike under their sleeves at the wrists and by rubbing it from above with the other hand, or by just moving the sleeve, the grass moves quickly towards the shoulder. The one-way travel of spikes in the respiratory tract is accomplished by the respiratory movements and cough.

Referred diffuse abdominal pain seems to be a characteristic clinical manifestation. The first patient complained of abdominal pain 6 days after the probable inhalation of the foreign body, and the second only 12 hours after the inhalation.

The roentgenologic findings of the chest are not specific and, therefore, it is not feasible to make a firm diagnosis on this basis. In any case, foreign bodies in the respiratory tract whose inhalation has been overlooked should not be considered as exceptional rarities. The most valuable help is a detailed medical history, which will usually mention a sudden attack of dyspnea, cough and wheezing respiration.

In both cases, shadows developed throughout the involved lobe of the lung and subsequently the inflorescence pointed at the chest wall in a subcutaneous position.—*James F. Martin, M.D.*

SCHNEIDER, LOUIS, and WIMPFHEIMER, FELIX. Multiple progressive calcific pleural plaque formation: a sign of silicatosis. *J.A.M.A.*, July 27, 1964, *189*, 328–330. (Address: Dr. Schneider, 600 W. 168th Street, New York 32, N. Y.)

Case histories and findings are given on 3 patients with disseminated calcific pleural plaque formation. Two of these patients gave histories of long exposure to asbestos. Literature review indicates that such lesions may be produced by occupational or non-occupational exposure to certain silicates, notably anthophyllite asbestos and tremolite talc.

This rare type of calcification is presumed to occur in the parietal pleura and tends to be bilateral. Solitary or confluent spots or figures may be seen. There is a predilection for the inner chest wall and may involve the basal and mediastinal portion of the pleura. Observation of the lesions over the course of many years shows slow progression in size.

Mechanical irritation has been proposed by other authors in the literature as being responsible for the pathologic changes.—*George A. Miller, M.D.*

KREEL, L., BLENDIS, L. M., and PIERCY, J. E. Pneumo-mediastinography by the trans-sternal method. *Clin. Radiol.*, July, 1964, *15*, 219–223. (From: Departments of Radiology and Endocrinology, New End Hospital, Hampstead, London, N.W. 3, England.)

In myasthenia gravis when a thymoma is present, it has been shown that the best treatment is radiation therapy followed by removal of the thymus causing the tumor. A number of thymic tumors may escape detection by conventional methods. Pneumomediastinography, however, is a well recognized diagnostic procedure. The methods described for inducing a pneumo-mediastinum include the trans-tracheal method and the suprasternal notch method. In view of the experience of one of the authors of this article (L. K.) with the transcoccygeal method of retroperitoneal air insufflation, the technically similar trans-sternal puncture was applied to pneumo-mediastinography. This trans-sternal method has been used both in the presence and absence of the manubriosternal joint.

The thymus increases in size from birth to puberty, and can be anything from 5 to 15 cm. in length. In adolescence the gland begins to decrease in size with fat replacement. The pleura moves across the anterior mediastinum towards the mid-line during inspiration. Therefore, the needle is always inserted in deep expiration and tomography is always done in deep inspiration.

Amytal 50 mg. is used as premedication half an hour prior to the procedure. A metal marker is placed over the manubriosternal joint, localized by palpation. A lateral roentgenogram is then taken centering on the marker. The subcutaneous tissue is infiltrated with a local anesthetic. A 19 or 20 gauge needle, approximately 4 cm. long, is inserted in expiration through the joint space in the mid-line, so that its tip lies 0.5 cm. deep to the posterior sternal margin. A control roentgenogram is then taken, and, if the position of the needle is satisfactory, the stilette is removed and a syringe and two-way tap are connected. A small quantity of saline (5 cc.) is then injected as a safety precaution against air embolism. Carbon dioxide is slowly injected. After the first 100 cc. of the gas has been injected, an additional lateral roentgenogram is taken to check on the space created in the anterior mediastinum. Then a total of 400 to 500 cc. of air is injected. Lateral and prone postero-anterior tomography of the anterior mediastinum is now carried out.

The thymus is well shown by this method, and the walls of the aorta, trachea and pulmonary arteries can be demonstrated. Where lesions of the trachea or bronchus are present (*e.g.*, bronchial adenoma), extension into the surrounding tissue could be shown. Intrathoracic parathyroid tumors could possibly also be demonstrated in this way.—*Samuel G. Henderson, M.D.*

ABDOMEN

Delahaye, R. P., Jolly, R., Laaban, J., and Bourisiquot, P. L'amibiase intestinale chronique (étude radiologique de 171 cas). (Chronic intestinal amebiasis: radiologic study of 171 cases.) *J. de radiol., d'électrol. et de méd. nucléaire*, Mar.-April, *45*, 117–132. (From: Services de Médicine générale et d'Electroradiologie de l'H. M. I. Dominique-Larrey à Versailles (Seine-et-Oise), et du Service d'Electroradiologie de l'H. M. I. du Val-de-Grâce, Paris, France.)

After a short survey of the clinical and topographic aspect, a detailed discussion of the fluoroscopic and roentgenographic findings of chronic amebiasis of the cecum, large bowel and rectum is presented.

The fluoroscopic findings include rapid progression of the barium fluid, especially through the ileocecal junction, and localized constant segmental spasm. The haustral anomalies are described in detail and illustrated by drawings and roentgenographic reproductions of some of the interesting cases. They consist of localized or multifocal spasms and very densely serrated haustrations with many variations in between. The same is true of the alterations in the caliber of the large bowel. The segmental, localized forms comprise by far the largest number, as compared to the generalized forms; the localized forms are observed mostly in the ascending and sigmoid colon, where areas of amebic changes are intermingled with normal segments. This is characteristic in almost all cases of amebic involvement.

The tumoral form of amebic pathology is also discussed and a few cases presented. The tumoral form of amebiasis gives rise to a longer area of constriction as compared to a neoplasm and the line of demarcation is not as sharp. The mucosal pattern is less disrupted and there is slight expansion of the narrowed segment of the large bowel during the flow of barium, whereas in case of cancer the narrowed lumen remains rigid. Also, obstruction is less complete in case of amebic tumors and, hence, less pain is precipitated during barium enema study. The therapeutic test with ematine is of further differential diagnostic value. In tuberculosis of the cecum, the terminal loops of the ileum are more often involved than in amebiasis. In circumscribed appendiceal abscess demonstration of an extrinsic pressure defect is helpful. Colonic syphilis and actinomycosis should also be ruled out in diagnosis of the tumor forms of amebiasis.—*Jirair N. Sarian, M.D.*

Brunton, F. J. Choristoma of the gall-bladder. *Clin. Radiol.*, July, 1964, *15*, 283–285. (From: Diagnostic X-ray Department, St. Mary's Hospital, London, W.2, England.)

Discussions on the differential diagnosis of polypoid lesions of the gallbladder rarely mention ectopic tissue. A case is here reported in which cholecystographic examination revealed an irregular polypoid filling defect arising from Hartmann's pouch. At operation a soft, mobile tumor could be felt in the gallbladder. On examining the removed organ, it was found that the tumor readily prolapsed into the neck of the gallbladder. On pathologic examination, in the infundibulum, there was a hamartomatous "tumor" composed of gallbladder and gastric type glands. The gastric glands contained both chief and parietal cells, and were of the type found in the fundus of the stomach.

Only 6 cases of polyps containing gastric glands have been reported previously. This case and 4 of the previously reported cases occurred in the region of the neck of the gallbladder. The Armed Forces Institute of Pathology classifies such a tumor as a "choristoma" of gastric glands. A choristoma (Greek-*choristos* separated+*oma* tumor) is defined as a mass of tissue histologically normal for an organ or part of the body other than the site at which it is located. All 8 of the polypoid lesions now reported in the literature composed of ectopic tissue have been found in the same region of the gallbladder. Cholesterol polyps, granulomas and adenomata are usually found in the middle third of the gallbladder. A discrete adenomyomatous tumor is usually sessile and is found at the fundus.—*Samuel G. Henderson, M.D.*

Haastert, S. Verträglichkeit und diagnostische Ergebnisse bei der klinischen Erprobung von Bilivistan (SH 419). (Tolerability and diagnostic results of the clinical trial of bilivistan [SH 419].) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Aug., 1964, *101*, 176–180. (Address: Röntgenabteilung der Med. und Chirurg. Poliklinik der Universität Köln, Köln 5, Germany.)

A new cholecystographic drug bilivistan (SH 419) was tested by the author because it was claimed to have a better tolerability and less side effects than similar drugs. Its greatest advantage was a simultaneous visualization of the bile ducts and the gallbladder. In a series of 53 cases, malaise and nausea were observed in 30 per cent and no reaction in 34 per cent. All other side effects were minimal and negligible. Only 50 cases could be diagnostically evaluated.

Roentgenograms were made at intervals of 15 minutes, and it was soon discovered that the bilivistan remained in the bile ducts longer than other media. Optimal filling of the gallbladder and the bile ducts was obtained 45 to 105 minutes after the intravenous injection of the drug.

The filling of the *bile ducts* was very good in 4 cases, good in 24, satisfactory in 10, and adequate in 9. Two cases had adequate visualization of the gallbladder but not of the bile ducts. In the remaining case, a preceding choledochoduodenostomy resulted in absence of the sphincter of Oddi effect with rapid emptying into the duodenum.

The visualization of the *gallbladder* was good in 35 patients but rather faint in 4; in the remaining 11 it was either poor or absent. Five of these had occlusion of the cystic duct, 5 others had a preceding cholecystectomy, and 1 a choledochoduodenostomy without cholecystectomy. In 5 patients the contrast material was simultaneously excreted by the kidneys and twice a hepatogram effect was obtained.

The author concludes that bilivistan is as well tolerated as biligrafin forte (50 per cent) and that the diagnostic results are excellent. A considerable advantage is the simultaneous visualization of the gallbladder and the bile ducts in roentgenograms made 90 minutes after the intravenous injection.—*Ernest Kraft, M.D.*

Elkington, S. G., and Bernstein, D. C. Percutaneous transhepatic cholangiography. *Brit. M. J.*, Sept. 19, 1964, *2*, 733–735. (Address: Dr. Elkington, Lately Medical Registrar, St. Thomas' Hospital, London, England.)

Percutaneous transhepatic cholangiography is useful when the serum bilirubin is greater than 2–3 mg./100 ml. and other methods are ineffective. It can demonstrate the dilated intrahepatic biliary system which develops in chronic extrahepatic obstruction. A negative result can remove the need for laparotomy in a patient with hepatocellular disease.

The authors use Kaplan's technique, a 15 cm. needle with a 1 mm. bore inserted under local anesthesia through a point immediately below the right costal margin and 3 or 4 cm. from the midline. The needle is advanced for 12 cm. into the liver at an angle of 45 degrees to the horizontal and inclined to the patient's right. Suction is applied and the needle slowly withdrawn until bile is obtained. As much bile is aspirated as possible and an appropriate contrast medium injected. Facilities for laparotomy must be readily available for surgical intervention is essential if the complications of hemorrhage or biliary peritonitis occur.

The authors state that at least 413 such investigations have been reported in the literature with risk of complication in the region of 4 to 6 per cent. Mortality from surgical exploration alone is from 3 to 10 per cent. The value of this investigation in selected cases of chronic obstructive jaundice should be more generally appreciated.—*A. H. Hall, M.D.*

GENITOURINARY SYSTEM

CRAVEN, J. D. Renal glomerular osteodystrophy. *Clin. Radiol.*, July, 1964, *15*, 210–218. (From: Diagnostic X-ray Department, University College Hospital, London, England.)

Roentgenologic changes which occur in the skeleton of patients with chronic renal failure are described under the term of "renal osteodystrophy." Such changes include: (1) dwarfism and retardation of growth; (2) rickets or osteomalacia; (3) osteitis fibrosa generalisata; and (4) osteosclerosis. These changes are a result of chronic glomerular failure, but identical changes can occur in long-standing steatorrhea, and as a late event in some cases of renal tubular failure, especially in the Fanconi syndrome. These alterations are a response to a deficiency of, or lack of response to normal amounts of, vitamin D, which has several causes. In steatorrhea there is a state mimicking chronic vitamin D deficiency, and in renal glomerular osteodystrophy an acquired resistance to the normal action of vitamin D. This vitamin is always curative if given in adequate dosage.

In the renal group, interest is being shown in the nature of the fundamental renal lesion, and the possibility of its treatment. Chronic pyelonephritis is the commonest renal lesion to produce renal glomerular osteodystrophy. Chronic glomerulonephritis and polycystic kidney disease are relatively uncommon causes. Vesico-ureteric reflux is frequently associated and should always be considered as a possible underlying cause.

The presenting symptoms include failure to grow, limb deformities, pain especially in the back and in the legs, weakness in the legs, urinary tract symptoms, and symptoms from uremia. The physical signs include dwarfism, deformities such as kyphosis, knock knee, bow legs, Harrison's sulcus, swellings of the costochondral junctions, pseudoclubbing of the fingers, bony tenderness and the physical signs of chronic uremia and anemia. The absence of hypertension early in the disease is characteristic.

The average serum calcium is low at 8.4 mg. per cent, the average serum phosphorus is raised at 5.6 mg. per cent, the average blood urea is raised at 167.0 mg. per cent, and the average serum bicarbonate is reduced at 14.7 mEq./L. There is an associated monochromic anemia, with average hemoglobin value of 65 per cent. The alkaline phosphatase is raised to an average of 48 King Armstrong units, and here, as in primary hyperparathyroidism, there is a close correlation between the presence of bone changes and the rise in alkaline phosphatase. Treatment with vitamin D in high dosage will produce a positive calcium balance and heal the rachitic change.

The bone changes of hyperparathyroidism are commonest. Next most common is osteosclerosis, especially in the lumbar and occasionally in the thoracic vertebral bodies. Rickets occurred in 5 of 20 patients, and acute osteomalacia was seen in 3 cases, with definite chronic osteomalacia in only 1 of the 20. There is still no adequate explanation for the appearance of one type of change rather than another, nor is there any explanation of the etiology or significance of the osteosclerosis. The excess of normal bone is paradoxical in the presence of conditions of bone removal and of production of uncalcified osteoid.—*Samuel G. Henderson, M.D.*

SCHENCKER, BERNARD. Drip infusion pyelography: indications and applications in urologic roentgen diagnosis. *Radiology*, July, 1964, *83*, 12–21. (From: Department of Radiology, Brooke General Hospital, Fort Sam Houston, Texas.)

By using a drip infusion technique with larger than usual quantities of contrast medium, the author obtained excellent nephrograms, pyelograms, cystograms and voiding urethrograms. Its principal use was as a secondary procedure when the standard excretory urograms were not diagnostic. The anatomic detail obtained makes retrograde pyelography unnecessary in most cases.

The excellence of the technique is achieved by virtue of hydration, diuresis and complete filling of the urinary tract. The recommended dosage is 1 cc. per pound of body weight of 5 per cent hypaque mixed with 1 cc. per pound of 5 per cent dextrose in water. The minimal adult dose is 150 cc. of each. It was used only in adults.

Various adaptations of this technique are presented, including nephrotomography and "wash out" studies.—*G. W. Pett, M.D.*

BLISS, B. P., CLARK, WILLIAM G. B., and SAUNDERS, MICHAEL. Recurring spontaneous rupture of the urinary bladder following subarachnoid haemorrhages. *Brit. J. Surg.*, June, 1964, *51*, 446–448. (From: Charing Cross Hospital, London, England.)

Recurrent spontaneous rupture of the bladder in the absence of urinary tract disease has been reported on four prior occasions in the literature. Details of a fifth case are presented by the authors in which both ruptures occurred following subarachnoid hemorrhages of the brain. The first rupture was on the posterior aspect of the bladder vault into the peritoneal cavity. The second rupture occurred at the left edge of the scar from the previous rupture.

It is postulated by the authors that neuromuscular incoordination at the bladder neck associated with some bladder retention was responsible for the ruptures which occurred at the weakest point of the bladder.—*George A. Miller, M.D.*

Vermooten, Vincent, and Schweinsberg, Michael. Radiographic estimation of the size of the prostate. *Radiology*, June, 1964, *82*, 1010–1015. (Address: 3607 Gaston Avenue, Dallas, Texas 75246.)

Roentgenographic and clinical methods for estimating the size of an enlarged prostate are often quite inaccurate.

The authors state that this information can be helpful in determining the surgical approach, and from their study of the various methods available made the following conclusions: (1) "elevation of the floor of the bladder" is significant only when it is accompanied by 'J'-shaped distal ureters on excretory urography; (2) urographic media when excreted frequently raise the urine specific gravity to 1.045 or 1.060, in contrast with normal urine with specific gravity of 1.015 to 1.020. These two urines do not readily mix; therefore, on excretory urography urine already in the bladder may give an incorrect first impression of a filling defect in the region of the prostate; (3) opaque cystography frequently obscures the "intravesical prostate"; (4) intravesical protrusion of the enlarged prostate is best observed in an air cystogram. A 45° oblique view is especially valuable.

Roentgenographic methods presently available do not permit accurate preoperative prediction of the size or weight of the prostatic enlargement. Particular caution should be exerted to avoid inaccurate interpretations from excretory cystograms or urethrocystograms.—*G. W. Pett, M.D.*

SKELETAL SYSTEM

Ferrand, J., Viallet, P., Chevrot, L., Chitour, M., and Djilali, G. La périostite albumineuse d'Ollier et Poncet: (étude angiographique segmentaire et abcessographique). (Albuminous periostitis of Ollier and Poncet [segmental angiographic studies and abcessography].) *J. de radiol., d'électrol. et de méd. nucléaire,* May, 1964, *45*, 209–214. (From: Chaire de Clinique électroradiologique [Alger]; Service Central d'Electroradiologie, Hôpital Sainte-Marguerite [Marseille]; and Chaire de Thérapeutique chirurgicale et orthopédique [Alger].)

Clinically, this entity is characterized by a painless collection, often enormous or monstrous, neglected since its insidious beginning. When punctured, a yellowish viscous fluid is withdrawn, similar to the white of an egg, which is Rivalta positive and contains a few supernatant droplets. The clinical history reveals long-forgotten episodes of staphylococcic osteomyelitis.

Roentgenologically, signs of old osteomyelo-periostitis can be seen, and also a soft tissue swelling adjacent to the bone lesion, which corresponds to the painless collection. This abscess is clearly visible in the soft tissues and confirmed by a puncture through which a synovial-like fluid is obtained, very albuminous and rich in gamma globulins. For the radiologist, these are the signs of albuminous periostitis of Ollier and Poncet.

The authors report a case of a 20 year old Algerian woman, who presented an enormous semi-ovoid mass in the posterior lower third of the thigh, which was a relapsing albuminous collection similar to the first one operated upon 5 years previously. The roentgenologic segmental investigation included plain roentgenography, tomography for bone study, phlebography with provoked stasis, arteriography, then abcessography. This last examination demonstrated the inside of the collection structure and showed the importance of fungous formations and free or adherent false membranes which exhibited an iodophilic property.

The albuminous collection behaves like a cystic tumor, displacing the femoral vascular vessels. The venous circulation, more easily influenced, is diminished. Increased arterial vascularization can be seen around the pseudocyst. All this helps to understand the progressive and unrelenting nature of the exudation.

A meticulous roentgenologic investigation greatly facilitates the surgical removal. Upon histologic examination, the nonsuppurative and nonspecific inflammatory character of the affection is demonstrated. The electrophoresis reveals a greatly increased concentration of gamma globulins, which may be explained by locally acquired immunity phenomena.—*H. P. Lévesque, M.D.*

Carbonara, Peter, and Alpert, Meyer. Hereditary osteo-onycho-dysplasia (HOOD). *Am. J. M. Sc.*, Aug., 1964, *248*, 139–151. (From: Departments of Orthopedic Surgery and Radiology, Columbia-Presbyterian Medical Center, New York, N. Y.)

Hereditary osteo-onycho-dysplasia (HOOD) is a syndrome affecting the nails, elbows, pelvis and knees. This term was first used by Roeckerath in 1951. The condition had formerly been called Turner's syndrome, or Osterreicher-Turner's syndrome. (Described by J. W. Turner, not to be confused with H. H. Turner, one of the first to describe gonadal dysgenesis.) It has also been called Fong's syndrome and Touraine's syndrome. In the early literature, this syndrome has been incompletely described, or inconstant features have been included in its descriptions.

The authors have obtained their data from 60 cases adequately documented in the literature since 1948, and 2 of their own cases. Diagnosis of HOOD was most commonly made when the patients were in the second and third decade, and because of the family history.

Males and females were equally affected and were Caucasian except for 1 Negro.

The most contant defects were the nail changes which affected 98 per cent of the cases and ranged from longitudinal ridging to complete anonychia. The defects were always symmetric and the thumb nails and great toe nails most severely involved.

Knee changes were noted in 92 per cent. The roentgenographic changes usually showed hypoplasia of the lateral femoral condyle, with flattening of the articular surface. Also noted were hypoplasia of the fibular head, dysplasia of the tibial plateau, and hypoplasia of the patella with subluxation or dislocation. In 11 per cent both patellae were absent.

The next most common deformity was in the elbows, with 90 per cent involvement. The roentgenograms generally showed prominence of the medial epicondyle of the humerus, hypoplasia of the capitellum and lateral epicondyle. Elongation and deformity of the radial head, with bowing of the shaft were seen, and when subluxed or dislocated, were associated with shortening of the ulna. Elbow defects were not always symmetric.

Iliac horns were present in 81 per cent, and were always symmetric, arising from the central area of the external iliac fossa. The bases were 2–5 cm. in diameter, and the lengths varied up to 4 cm. Since these horns do not occur in any other condition, they are pathognomonic of HOOD.

The condition is transmitted as a mendelian dominant trait, and it is believed that the transmission is mediated by a single mutant autosomal gene.

The complete tetralogy of nail, knee, elbow and pelvic changes was seen in 63 per cent of the patients. —*James R. Knapp, M.D.*

RADIOISOTOPES

Entzian, W., Aronow, S., Soloway, A. H., and Sweet, W. H. A preliminary evaluation of F^{18}-labeled tetrafluoroborate as a scanning agent for intracranial tumors. *J. Nuclear Med.*, July, 1964, *5*, 542–550. (From: Neurochirurgische Universitäts Klinik Bonn, Germany, and Massachusetts General Hospital, Boston, Mass.)

One of the principal methods employed for radioisotopic localization of brain tumors is positron coincidence scanning. The positron-emitting isotopes which have been used in man are arsenic 72 and arsenic 74 as sodium arsenate. The latter has a relatively long half-life, 17.5 days. Copper 64 chelates have also been employed. With scintillation camera imaging devices, the 12.8 hour half-life of copper 64 is not a drawback. Fluorine 18 is a pure positron emitter with a 112 minute half-life, and the whole body dose to the patient receiving 1 mc of fluorine 18 is 0.05 rads compared to 0.42 rads for the usual dose, 3.0 mc of copper 64, and 1.8 rads for the diagnostic dose of 1.3 mc of arsenic 74.

Askenasy has shown localization in intracranial lesions of the BF_4 anion. The physical advantages of fluorine 18 and the possibility of labelling organic compounds suggested good properties as a tumor-localizing agent.

Distribution studies show a favorable ratio of tracer between a subcutaneously implanted tumor and brain in mice. Toxicity studies showed the anion to be well tolerated without any apparent acute toxic symptoms. There were no sequelae over a period of 6 weeks.

Results in scanning a small group of patients with surgically demonstrated lesions are given, and the results with fluorine 18 tracer are compared with those with copper 64 or arsenic 74. The new tracer appears worthy of further investigation.—*Frederick J. Bonte, M.D.*

SUBJECT INDEX TO VOLUME 92

(ab) = abstract (E) = editorial

- ABDOMINAL aorta, thrombotic occlusion of; seven year angiographic study of case, 877
  - aortic aneurysms, gastrointestinal manifestations of, 1110
  - aortography, and peripheral arteriography with single arm pressure injection, intravenous angiocardiography, 893
  - disease, specialized roentgen diagnostic technics in investigation of: annual oration in memory of Clarence Elton Hufford, 1893–1958 (ab), 961
  - pain, disk infections in (ab), 721
- ABRAHAM LINCOLN and Marfan syndrome (ab), 246
- ACNE LESIONS, calcified, roentgenographic appearance of (ab), 242
- ACOUSTIC NEURINOMA, percutaneous retrograde brachial angiography in diagnosis of; results in 4 cases, 829
- ADAMANTINOMA, so-called, of long bones: report of case with massive pulmonary metasis (ab), 247
- ADENOPATHIES, alterations of lymphatic ducts concomitant with neoplastic and systemic (ab), 724
- ADOLESCENTS exposed in utero to atomic bomb, Nagasaki, Japan, study of (ab), 1221
- AGLOSSIA, congenital (ab), 1434
- AIR-GAP TECHNIQUE; and improvement by anteroposterior positioning for chest roentgenography, 688
- AIR TRAPPING, pulmonary insufficiency and fluorodensimetry, 501
- ALBUMINOUS periostitis of Oller and Poncet (segmental angiographic studies and abscessography) (ab), 1441
- ALVEOLAR PROTEINOSIS, pulmonary; roentgenologic analysis, 571
- AMEBIASIS, chronic intestinal: radiologic study of 171 cases (ab), 1438
- AMERICAN ROENTGEN RAY SOCIETY, instruction courses of sixty-fifth annual meeting of (E), 455
  - section on instruction, 457
  - sixty-fifth annual meeting of (E), 218, 1410
  - sixty-fifth annual meeting of: preliminary program, 225
- AMOEBIC HEPATITIS, x-ray films in diagnosis of chronic (ab), 482
- AMYENTERIC ACHALASIA of esophagus (ab), 1212
- ANEMIA, syndrome of benign thymoma and primary aregenerative: analysis of forty-three cases (ab), 960
- ANEURYSM, false (ab), 1219
  - of vein of Galen; case report and review of literature, 756
- ANEURYSMAL MALFORMATION of vertebral and basilar arteries causing cranial nerve involvement, case of, 836
- ANEURYSMS of thoracic aorta, arteriosclerotic; intravenous and selective visualization, 1353
- ANGIOCARDIOGRAPHIC DEMONSTRATION of obstructing left atrial membrane; cor triatriatum 669
- ANGIOCARDIOGRAPHY, selective, intramural deposition of contrast agent during (ab), 492
- ANGIOCONRAY, severe reaction to (ab), 970
- ANGIOGRAM, pulmonary thromboembolism—case for pulmonary (E), 693
- ANGIOGRAPHIC artefacts which simulate or mask abnormality, 907
  - demonstration of bilateral carotid body tumors, 850
  - diagnosis of intralobar pulmonary sequestration, 540
  - diagnosis, ophthalmic arterial complex in (ab), 1433
  - features of unilateral hydrocephalus of obstructive nature, 792
  - media, red cell aggregation from concentrated saline and (ab), 970
  - research, arterial simulator for, 935
- ANGIOGRAPHY in acute renal failure (ab), 486
  - in diagnosis of suprasellar tumors, evaluation of, 311
  - in localization of parathyroid tumors, adaptability of selective (ab), 1212
  - programmed, 939
- ANGIOMEGALIES, portal (ab), 1218
- ANTERIOR cerebral artery, unilateral intracranial hematoma without shift of, 786
  - choroidal artery in intracranial lesions, changes in, 776
- ANTEROPOSTERIOR POSITIONING for chest roentgenography, and improvement by; air-gap technique, 688
- ANTICHOLINERGIC DRUG in patient with ulcerative colitis, megacolon associated with administration of: report of case (ab), 715
- AORTIC ATRESIA without significant hypoplasia of ascending aorta, 888
- AORTOGRAPHY for evaluation of renal artery lesions in renovascular hypertension, modification and extension of, 321
- ARCHAEOLOGY with x-ray apparatus (ab), 737
- ARMED FORCES INSTITUTE OF PATHOLOGY, 475
- ARTERIAL occlusion, acute, with streptokinase, thrombolytic treatment of (ab), 1219
  - simulator for angiographic research, 935
- ARTERIOGRAPHY and arthrography in pigmented villonodular synovitis of knee, 1322
  - coronary; some observations on technique and interpretation, 872

  evolution of cerebral (E), 948
  in study of renal hypertension, upright renal (ab), 486
  percutaneous noncatheter left brachial renal, 315
  percutaneous transfemoral; analysis of complications in 1,000 consecutive cases, 918
  serial, in vascular insufficiency of loser extremities (ab), 721
ARTERIOSCLEROSIS and myocardial infarction, calcification in coronary arteries and its relationship to, 865
ARTERIOSCLEROTIC ANEURYSMS of thoracic aorta; intravenous and selective visualization, 1353
ARTERIOVENOUS FISTULA, congenital intrarenal (ab), 486
ARTERITIS, giant cell (cranial); case with angiographic abnormalities, 769
ARTHROGRAPHY and arteriography in pigmented villonodular synovitis of knee, 1322
ARTHROPATHY, diabetic: contribution to its study (ab), 1218
ASCARIASIS of biliary pathways and digestive tract (ab), 1215
ASPLENIA: report of case diagnosed antemortem on basis of roentgenograms (ab), 484
ATHEROSCLEROTIC OCCLUSION of common carotid arteries, bilateral (ab), 721
ATOMIC BOMB, Nagasaki, Japan, study of adolescents exposed in utero to (ab), 1221
ATRIAL membrane, angiocardiographic demonstration of obstructing left; cor triatriatum, 669
  septal defect observed during 25 years; clinical and pathological data, with special reference to heart sounds and murmurs, case of (ab), 244
AUDITORY CANAL, normal internal, 1232
AUTOFLUOROGRAPHY of thyroid gland by means of image amplification (ab), 734
AUTORADIOGRAPHIC analysis of uptake of tritiated thymidine and S^{35} cystine by cultured human cervical explants undergoing metaplasia (ab), 733
AXIAL TRANSVERSE ENCEPHALOGRAPHY, 441
AZOTEMIC PATIENT, excretory urography in: revised technique (ab), 716

BARIUM, nonfatal intravasation of, 1169
  sulfate as bronchographic contrast medium, further experience with, 595
("BATTERED BABY" SYNDROME), multiple epiphysial injuries in babies (ab), 248
(BAUHIN'S VALVE), radiologic anatomy of ileocecal valve (ab), 481
BETA IRRADIATION, intraluminal, in leukoplakia, constricting male urethra (ab), 250
BICENTENNIAL of medical education in U.S.A., 699
BILIARY PATHWAYS and digestive tract, ascariasis of (ab), 215
BILIVISTAN (SH 419), tolerability and diagnostic results of clinical trail of (ab), 1439
BIRDSALL, EDGAR, M.D., 1876–1964 (E), 456
BLADDER, hemangioma of, 375
BOOK REVIEWS
  Elsevier's Medical Dictionary: in Five Languages, English-American, French, Italian, Spanish and German. Compiled by A. Sliosberg, 232
  The Lower Esophageal Vestibular Complex. By Costantino Zaino, Maxwell H. Poppel, Harold G. Jacobson, and Harold Lepow, 477
  World Directory of Medical Schools. Third edition. World Health Organization, Geneva, 477
  Gastroenterology. Volume II. By Henry L. Bockus, 702
  Une Radiographie du Crâne. Savoir Interpréter. By Lucien Appel and Jacques Simon, 702
  Year Book of Radiology: 1963–1964 Series. Radiologic Diagnosis. Edited by John Floyd Holt and Walter M. Whitehouse. Radiation Therapy. Edited by Harold W. Jacox and Morton M. Kligerman, 1203
  An Atlas of Vascular Rings and Related Malformations of the Aortic Arch System. By James R. Stewart, Owings W. Kincaid, and Jesse E. Edwards, 1203
  Radiologia Infortunistica della Colonna Vertebrale: Patologia Traumatica e Medicina Legale. By Lorenzo Giuntoli, and Bruno Guareschi, 1428
BONE changes following radiation therapy for malignant lesions in region of pelvis (ab), 732
  chondrosarcoma of (ab), 968
  desmoplastic fibroma of: report of two cases (ab), 719
  giant-cell tumor of: analysis and follow-up study of forty-one cases observed at Massachusetts General Hospital between 1925 and 1960 (ab), 247
  grafting, treatment by: osteochondritis dissecans (ab), 1217
  island, 1301
  lesions, use of strontium-85 for detection of (ab), 1223
  marrow smears, recognition and clinical importance of thorium dioxide (thorotrast) deposits in human unstained (ab), 733
  marrow transplantation for acute accidental irradiation injury, value of (ab), 254
  metastasis by photoscanning with strontium 85, diagnosis of (ab), 735
  mycotic infections of (ab), 969
  osteosarcoma of (ab), 247
  sarcomas following radiation therapy (ab), 731
  syphilis, report on certain recently observed cases of early congenital (ab), 487
BRACHIAL angiography, percutaneous retrograde,

  in diagnosis of acoustic neurinoma; results in 4 cases, 829
  renal arteriography, percutaneous noncatheter left, 315
BRAIN abscess by roentgenologic demonstration of an air-fluid level, preoperative diagnosis of; report of two cases, 844
  scanning, contour, with iodine and mercury compounds for detection of intracranial tumors, 177
  scans in management of suspected intracranial lesions, value of (ab), 734
  tumors, simulated, with gallium 68-EDTA, sensitivity of positron scintillation camera for detecting, 174
BREAST cancer, mammography as routine screening examination for detecting, 14
  cancer, should more radical treatment be attempted in? Caldwell Lecture, 1963, 3
  disease, calcification in: mammographic-pathologic correlation, 29
  roentgenographic diagnosis (E), 221
  therapeutic dilemma, carcinoma of. Gordon Richards Memorial Lecture—1964 (ab), 727
BRONCHIAL CARCINOID with carcinoid syndrome, metastasizing (ab), 244
BRONCHIOLAR DILATATION associated with muscular hyperplasia: polycystic lung; emphasis on roentgenologic findings, 513
BRONCHOGENIC CARCINOMA, clinical studies of combined chemotherapy and irradiation in, 88
  preoperative irradiation in, 77
BRONCHOGRAPHIC agent hytrast, study of clinical and pathologic reaction to, 615
  contrast medium, further experience with barium sulfate as, 595
BRONCHOGRAPHY, transcricothyroid, some observations on, 578
  using radiopaque catheter, selective transtracheal, 591
BRONCHOPULMONARY SEQUESTRATION, intralobar; review of literature and report of 2 cases, 547
Brucellosis with prolonged illness due to suppuration of liver and spleen, host-parasite relationship in human (ab), 492

CA 47, use of whole body counter in turnover studies with (ab), 1222
CALCIFIC PLEURAL PLAQUE FORMATION, multiple progressive: sign of silicatosis (ab), 1437
CALCIFICATION in breast disease: mammographic-pathologic correlation, 29
  in coronary arteries and its relationship to arteriosclerosis and myocardial infarction, 865
  in intracranial metastases, 1249
  in portal venous system: unusual radiologic sign of portal venous thrombosis (ab), 491
CALCIUM in biological tissues at microlevel, use of x-rays in detection of (ab), 973
CALDWELL LECTURE, 1963. Should more radical treatment be attempted in breast cancer?, 3
CAMPTODACTYLY, 1312
CANCER, 24 hour I^{131} uptake and conversion ratio in patients with, 192
  lung: detection by use of electrical skin resistance method (ab), 713
  of eyelid, radium therapy in (ab), 1222
CANDIDA ESOPHAGITIS, roentgenologic symptomatology of (ab), 245
CANINE CINEPORTOGRAPHY, 1139
CARBON DIOXIDE, double contrast barium cystography utilizing, 365
CARCINOID, metastasizing bronchial, with carcinoid syndrome (ab), 244
CARCINOMA, bronchogenic, clinical studies of combined chemotherapy and irradiation in inoperable, 88
  bronchogenic, preoperative irradiation in, 77
  of breast, therapeutic dilemma. Gordon Richards Memorial Lecture—1964 (ab), 727
  of esophagus: cobalt 60 teletherapy; experience and comparison with surgical results, 67
  of lung, environmental influences and (ab), 713
  of maxillary antrum, intracavitary cobalt 60 beads in treatment of, 55
  of skin, combination roentgen injuries after contact therapy of (ab), 729
  of tongue, complete oral reconstruction in conjunction with cobalt 60 teletherapy for; case report, 59
  of tongue in identical twins, 213
  of uterine cervix, urologic complications following irradiation of (ab), 251
  preoperative irradiation in bronchogenic, 77
  roentgen, 20 years after irradiation of thoracic wall sarcoma (ab), 729
CARCINOMAS, squamous cell, of larynx, evaluation of clinical usefulness of roentgenologic findings in, 43
CARDIAC prominence, posterior, in Ebstein's anomaly simulating mitral disease, 1331
  sarcoma, primary, successful sterilization of, by supervoltage radiation therapy, 942
CARDIOPATHIES, first observations on morphologic alterations of thoracic duct in some (ab), 724
CARDIOVASCULAR SYSTEM, Ellis-van Creveld syndrome: report of one case associated with abiotrophy of elastic tissue in (ab), 726
CAROTID body tumour in neck of newborn infant, malignant (ab), 712
  tumors, angiographic demonstration of bilateral, 850
CELIAC AND SUPERIOR MESENTERIC ANGIOGRAPHY, selective, indications for; experience with 128 cases, 1100

Cephalometrics, verticosubmental roentgenogram in (ab), 1212
Cerebellar hemispheres, frontal pneumostratigraphy of (ab), 1432
Cerebellum, generalized osteosclerosis secondary to metastatic medulloblastoma of (ab), 969
Cerebral arteriography, evolution of (E), 948
  hemispheres (medullary veins), veins of white matter of; diagnostic importance in carotid angiography, 739
Cervical cancers to irradiation by routine histological methods, assessment of response of (ab), 974
  discography, fallacy of; report of 50 cases in normal subjects (ab), 959
  rib, poststenotic dilatation (aneurysm) of subclavian artery associated with; report of two cases, 1328
  spine, incidence of acquired and congenital fusions in, 1255
  spondylosis, natural history and prognosis of (ab), 248
  spondylotic myelopathy studied by air myelography, 1260
Cesium 137 teletheraphy, preliminary observations in (ab), 975
Chemodectoma, retroperitoneal and mediastinal; report of case and review of literature, 1029
Chemotherapy and irradiation, combined, in inoperable bronchogenic carcinoma, clinical studies of, 88
Chest radiograph, diagnosis of dehydration, acidosis and gastroenteritis in infants from (ab), 1436
  roentgenography, and improvement by anteroposterior positioning for, air-gap technique, 688
Childhood, densimetry of bone in (ab), 720
Children, visceral injuries in battered (ab), 972
Cholangiography, percutaneous transhepatic (ab), 1439
  percutaneous transhepatic; preliminary report describing new approach to examination, 1131
  transhepatic (ab), 962
Cholecystographic agents, oral, experimental investigation of nephrotoxic effects of (ab), 483
Cholecystography, accelerated (ab), 1215
  inclined lateral decubitus position for, 1128
Cholecystokinin, studies on: its mode of action on gallbladder contraction (ab), 482
Chondrodystrophia calcificans congenita, case of (ab), 966
Chondrosarcoma, mesenchymal (ab), 968
  of bone (ab), 968
  of foot skeleton (ab), 731
Choristoma of gallbladder (ab), 1438
Chromophobe adenomas of pituitary gland, 160
Chyluria: lymphatic-urinary fistula demonstrated by lymphangiography (ab), 972
Cinecholangiography an aid in interpretation of T-tube cholangiogram deformities (ab), 962
Cinefluorographic advances, recent, in palatopharyngeal roentgenography, 977
Cinefluorography, physical factors in; exposure meter and phantom materials, 1188
  relative patient dose for 35 mm. and 16 mm. camera systems, 1192
Cineportography, canine, 1139
Cineradiography of esophageal varices (ab), 959
Cineroentgenographic examination of thoracic duct in man (ab), 723
Cineroentgenography of hollow muscular viscera, combined pressure tracings and, 986
Cine venography of lower extremity—preliminary report (ab), 722
Cirrhosis, intrahepatic portal thromboses following portocaval anastomosis for (ab), 491
Clarence Elton Hufford, 1893–1958, annual oration in memory of: specialized roentgen diagnostic technics in investigation of abdominal disease (ab), 961
Clavicle, three cases of osteolysis of acromial end of. Osteolysis after contusion (ab), 1216
Cleido-cranial dysostosis, hereditary: with note on anomaly of cementum (ab), 965
Clinical therapeutic radiology, radiation cataract in (ab), 976
Cobalt 60 beads, intracavitary, in treatment of carcinoma of maxillary antrum, 55
  teletherapy: carcinoma of esophagus; experience and comparison with surgical results, 67
  teletherapy for carcinoma of tongue, complete oral reconstruction in conjunction with; case report, 59
  teletherapy unit, double headed, 202
Colloidal zirconyl phosphate P^{32} in treatment of chronic leukemia and lymphomas (ab), 253
Colon, barium or air for examination of (E), 1200
  meandering, mesenteric artery of, 1088
  with spasmolytic drug given by rectum, radiologic examination of (ab), 481
Common carotid arteries, bilateral atherosclerotic occlusion of (ab), 721
  ventricle with inversion of infundibulum, roentgenographic features of; corrected transposition with rudimentary left ventricle, 652
(Conray-400), clinical evaluation of sodium iothalamate 66.8 per cent, 676
  iodine content 40 per cent; note on intravenous pyelography with 66.8 per cent sodium iothalamate, 267
Contrast agent during selective angiocardiography, intramural deposition of (ab), 492

examination of larynx and pharynx; inspiratory phonation, 40
media, influence of low molecular weight dextran on vascular and intravascular responses to, 682
COOLEY'S DISEASE in adult, considerations on radiographic aspect of (ab), 1218
CORONARY arteries, calcification in, and its relationship to arteriosclerosis and myocardial infarction, 865
arteriography; some observations on technique and interpretation, 872
heart disease, triolein-I^{131} tolerance in patients with (ab), 732
COR TRIATRIATUM; angiocardiographic demonstration of obstructing left atrial membrane, 669
CRANIOPAGUS TWINS, radiological examination of two pairs of (ab), 726
CUTANEOMANDIBULAR polyoncosis, hereditary: syndrome of myriad basal-cell nevi of skin, mandibular cysts, and inconstant skeletal anomalies (ab), 965
CYCLOPHOSPHAMIDE, congenital anomalies probably induced by (ab), 1224
CYSTINOSIS in 2 year old child, De Toni-Debré-Fanconi syndrome without demonstrable (ab), 1220
CYSTOGRAPHY, double contrast barium, utilizing carbon dioxide, 365

DECOMPRESSION SICKNESS, pulmonary changes in: some observations on compressed air workers at Clyde tunnel (ab), 243
DENSIMETRY of bone in childhood (ab), 720
DENTAL LESIONS in chronic fluorosis: origin of Sahara fluorine and its aggressiveness (ab), 1432
DERMATOMYOSITIS, acute, presenting with pulmonary manifestations (ab), 960
DE TONI-DEBRÉ-FANCONI SYNDROME without demonstrable cystinosis in 2 year old child (ab), 1220
DEXTRAN, low molecular weight, on vascular and intravascular responses to contrast media, influence of, 682
DIABETIC ARTHROPATHY: contribution to its study (ab), 1218
DIAGNOSTIC ROENTGEN-RAY DOSES, weighted gonadal, in teaching hospital with comments on x-ray dosages to general population of United States, 404
DIGESTIVE TRACT, ascariasis of biliary pathways and (ab), 1215
Hodgkin's disease and lymphosarcoma of (ab), 730
DISCOGRAPHY, lumbar (ab), 249
DISK INFECTIONS and abdominal pain (ab), 721
intervertebral, tumors of spine and their relationship to; roentgenographic-anatomic correlation, 1279
DISKOGRAPHY in radiology as based on 2,320 cases, place of, 1275
DIVERTICULOSIS, small intestinal, 1048
DUODENAL ULCER, stasis gastric ulcer: complication of (ab), 245
DUODENUM with controllable permanent magnets, recent advances in removal of magnetic foreign bodies from esophagus, stomach and, 1021
DYSPLASIA, ophthalmo-mandibulo-melic: hereditary syndrome (ab), 965

EBSTEIN'S ANOMALY of tricuspid valve; angiocardiographic considerations, 1338
simulating mitral disease, posterior cardiac prominence in, 1331
EDEMA, pulmonary: correlation of roentgenologic appearance and abnormal physiology, 495
EDGAR BIRDSALL, M.D., 1876–1964 (E), 456
EDITORIALS
The Sixty-fifth Annual Meeting of the American Roentgen Ray Society, 218
Breast Roentgenographic Diagnosis, 221
John D. Reeves, Jr., 1924–1964, 223
Preliminary Program: Sixty-fifth Annual Meeting of the American Roentgen Ray Society, 225
Instruction Courses of the Sixty-fifth Annual Meeting of the American Roentgen Ray Society, 455
Edgar Birdsall, M.D., 1876–1964, 456
Pulmonary Thromboembolism—the Case for the Pulmonary Angiogram, 693
The Evolution of Cerebral Arteriography, 948
Barium or Air for Examination of Colon, 1200
The Sixty-fifth Annual Meeting of the American Roentgen Ray Society, 1410
ELBOW JOINT, congenital (development) abnormalities of (ab), 488
ELECTROLYTE DEPLETION SYNDROME associated with villous adenomas of rectosigmoid, 1123
ELEVENTH INTERNATIONAL CONGRESS OF RADIOLOGY, 953
ELLIS-VAN CREVELD SYNDROME: report of one case associated with abiotrophy of elastic tissue in cardiovascular system (ab), 726
EMPHYSEMA, pulmonary, assessment of (ab), 712
ENCEPHALOGRAPHY, axial transverse, 441
ENDOCARDIAL FIBROELASTOSIS (ab), 961
ENDOCRINE OPHTHALMOPATHY: diagnostic and therapeutic problems (ab), 728
ENDOMETRIOSIS of ureter; rare manifestation of common disease, 351
ENVIRONMENTAL INFLUENCES and carcinoma of lung (ab), 713
EPIPHYSIAL INJURIES, multiple, in babies ("battered baby" syndrome) (ab), 248
ESOPHAGEAL mucosa, transmigration or prolapse of, 1017
reflux, 939

  - strictures, congenital, 994
  - varices, cineradiography of (ab), 959
  - varices in superior vena caval obstruction, 1000
- ESOPHAGUS, amyenteric achalasia of (ab), 1212
  - carcinoma of: cobalt 60 teletherapy; experience and comparison with surgical results, 67
  - stomach and duodenum with controllable permanent magnets, recent advances in removal of magnetic foreign bodies from, 1021
- ESTHESIONEUROEPITHELIOMA, olfactory: experiences in management of rare intranasal malignant neoplasm (ab), 241
- ETHIODOL following lymphangiography, body distribution of (ab), 972
- EUSTACHIAN TUBE obstruction and radiation therapy (ab), 975
- EWING'S SARCOMA (ab), 730
- EXCRETORY UROGRAPHY, double dose contrast medium in (ab), 717
  - in azotemic patient: revised technique (ab), 716

- F^{18}-labeled tetrafluoroborate as scanning agent for intracranial tumors, preliminary evaluation of (ab), 1442
- FAIRBANK'S DISEASE: rare familial systemic affection of skeleton (ab), 488
- FALX HEMATOMA, interhemispheric subdural or, 1252
- FEMORAL EPIPHYSIS, upper, early diagnosis of slipping of (ab), 967
- FEMUR, idiopathic avascular necrosis of head of: study of fifty-two cases (ab), 719
- FETAL LOBULATION of kidneys, 273
- FETUS, transverse presentation of (ab), 485
- FIBROUS DYSPLASIA, malignant transformation of (ab), 247
- FIRST A RADIOLOGIST; President's address, 1227
- FLUORODENSIMETRY, air trapping, pulmonary insufficiency and, 501
- FLUOROSIS, chronic, dental lesions in: origin of Sahara fluorine and its aggressiveness (ab), 1432
- FONG'S LESION—iliac horns with arthrodysplasia and dystrophy of nails: review of literature and report of family (ab), 967
- FORAMEN OF MORGAGNI simulating left ventricular hypertrophy, herniation of transverse colon through; report of case, 1013
- FOREIGN BODY, unusual (ab), 1221
- FRACTURES of first ribs, ununited bilateral fatigue: case report and review of literature (ab), 487
- FREQUENCY RESPONSE characteristics of x-ray films and screens, 426
- FUSIONS, acquired and congenital, in cervical spine, incidence of, 1255

- GALLBLADDER, choristoma of (ab), 1438
  - contraction, studies on cholecystokinin: its mode of action on (ab), 482
- GALLIUM-68 as scanning agent for intracranial lesions (ab), 734
  - -EDTA, sensitivity of positron scintillation camera for detecting simulated brain tumors with, 174
- GAMMA RAY fogging, uniform, on diagnostic usefulness of commercial x-ray film, effect of (ab), 727
- GAS ABSCESS within leiomyoma of uterus, 400
- GASTRIC radiology, high voltage and (ab), 714
  - tridermal teratoma in infant (ab), 714
  - ulcer, stasis: complication of duodenal ulcer (ab), 245
- GASTRODUODENAL LEIOMYOSARCOMAS: report of clinical, roentgenologic, and pathologic features of three surgical cases (ab), 246
- GASTROINTESTINAL lesions, microangiographic study of, 1173
  - manifestations of abdominal aortic aneurysms, 1110
- GASTROPATHY, hypertrophic hypersecretory: analysis of 15 cases and review of pertinent literature (ab), 1214
- GAUCHER'S DISEASE, osseous, in early childhood: report of case with extensive bone changes and pathological fracture without splenomegaly (ab), 488
- GENITAL CANCER, lymphography in (ab), 725
- GERMINAL TESTICULAR TUMORS, 152
- GIANT cell (cranial) arteritis; case with angiographic abnormalities, 769
  - tumor, "benign," of bone with solitary pulmonary metastasis: case report (ab), 968
- GLOMERULAR FILTRATION RATE, I^{125} allyl inulin for determination of, 302
- GLOMUS TUMOR of stomach; case report, 1026
- GONADAL dysgenesis, skeletal abnormalities in (ab), 718
  - weighted, diagnostic roentgen-ray doses in teaching hospital with comments on x-ray dosages to general population of United States, 404
- GORDON RICHARDS MEMORIAL LECTURE—1964. Carcinoma of breast, therapeutic dilemma (ab), 727
- GRANULOMA, rust, of lung: contribution to corroding process of metallic splinters in body (ab), 1434
- GRANULOMAS, thorotrast, 584
- GRASS INFLORESCENCES inhaled through lung and chest wall, spontaneous elimination of (ab), 1437
- GREAT VESSELS, roentgenographic features of congenital corrected transposition of; comparative study of 33 cases with roentgenographic classification based on associated malformations and hemodynamic states, 623
- GRID IRRADIATION in telecesium therapy (ab), 250

HAEMOPTYSIS, routine radiography for (ab), 242
HAMARTOMA of spleen; angiographic observations, 1150
HAMOLSKY TEST, in rat after administration of anticoagulant substances (heparin and 4-hydroxycoumarin), modifications of (ab), 1443
HAND, right, accidental, occupational radiation injury to (ab), 729
HEART, further experiments on long term survivors after circulatory bypass of right side of (ab), 1213
  malformed, visceral heterotaxy with: similar findings in child and puppy (ab), 961
HEMANGIOMA of bladder, 375
HEMATOMA, unilateral intracranial without shift of anterior cerebral artery, 786
HEMOGLOBIN C DISEASE: quantitative determination of iron kinetics and hemoglobin synthesis (ab), 726
HEMOSIDEROSIS, nodular aspirational pulmonary, 561
HEPATITIS, chronic amoebic, x-ray films in diagnosis of (ab), 482
HERNIATION of transverse colon through foramen of Morgagni simulating left ventricular hypertrophy; report of case, 1013
HERPES ZOSTER in reticuloses, disseminated, 116
HIATAL HERNIAS, natural evolution of (ab), 714
HIGH energy radiation therapy of pulmonary neoplasms, complications and after effects of (ab), 974
  voltage radiation: adjunct to surgery in large extrinsic malignancies of larynx (ab), 249
HIP JOINT, bilateral anomalous development of: post mortem study of human fetus, twenty-six weeks old (ab), 720
HISTOPLASMOSIS; radiogical features of benign pulmonary form in South Africa (ab), 960
HODGKIN'S DISEASE and lymphosarcoma of digestive tract (ab), 730
  of lung; roentgenologic-pathologic correlation, 96
HOLLOW MUSCULAR VISCERA, combined pressure tracings and cineroentgenography of, 986
HUFFORD, CLARENCE ELTON, 1893–1958, annual oration in memory of: specialized roentgen diagnostic technics in investigation of abdominal disease (ab), 961
HUMAN PHANTOM: improved for teaching and testing, 215
HYDROCEPHALUS, unilateral, of obstructive nature, angiographic features of, 792
HYPERCALCURIC RICKETS associated with renal tubular damage (ab), 964
HYPERPARATHYROIDISM, occult, and rapid calcium infusion test (ab), 489
HYPERTHYROIDISM in seventy-three children and adolescents, late results of I^{131} treatment of (ab), 735
HYPOGAMMAGLOBULINAEMIA, congenital, pneumocystis Carinii pneumonia and (ab), 1435
HYPOPHOSPHATEMIA, osteopathy in: clinical and radiologic findings (ab), 966
HYPOPLASIA of ascending aorta, aortic atresia without significant, 888
HYTRAST, study of clinical and pathologic reaction to bronchographic agent, 615

I^{124} for positron scanning of neoplasms, labeling of human globulin with (ab), 735
I^{125} allyl inulin for determination of glomerular filtration rate, 302
I^{131} 24 hour uptake and conversion ratio in patients with cancer, 192
  -cholografin and IHSA heart scan, measurement of pericardial fluid correlated with (ab), 252
  treatment of hyperthryoidism in seventy-three children and adolescents, late results of (ab), 735
  T_3 test, resin sponge modification of (ab), 251
IDIOPATHIC AVASCULAR NECROSIS of head of femur: study of fifty-two cases (ab), 719
IHSA heart scan, measurement of pericardial fluid correlated with I^{131}-cholografin and (ab), 252
ILEOCECAL VALVE (Bauhin's valve), radiologic anatomy of (ab), 481
IMAGE amplification, autofluorography of thyroid gland by means of (ab), 734
  intensifier television systems, performance evaluation of, 418
INFANCY and childhood, pneumoperitoneum in (ab), 484
INFANT, newborn, malignant carotid body tumor in neck of (ab), 712
INFANTILE and adolescent adeno-broncho-parenchymal lesions following primary pulmonary tuberculous infection, clinical radiologic features of (ab), 959
INFANTS from chest radiography, diagnosis of dehydration, acidosis, and gastroenteritis in (ab), 1436
INFERIOR VENA CAVA in late pregnancy, studies of (ab), 484
INFUNDIBULUM, roentgenographic features of common ventricle with inversion of; corrected transposition with rudimentary left ventricle, 652
INFUSION PYELOGRAPHY, 1391
INSPIRATORY PHONATION; contrast examination of larynx and pharynx, 40
INSTRUCTION, American Roentgen Ray Society section on, 457
  courses of sixty-fifth annual meeting of American Roentgen Ray Society (E), 455
INTERHEMISPHERIC SUBDURAL or falx hematoma, 1252
INTERNAL auditory canal, normal, 1232
  carotid artery in intracranial lesions other than

  - meningiomas, visualization of tentorial branches of (ab), 1433
- INTESTINAL evacuation study: new roentgenologic technique (ab), 714
  - intramural, hemorrhage, 1061
  - knot syndrome, 1042
- INTRACRANIAL lesions, changes in anterior choroidal artery in, 776
  - lesions, gallium-68 as scanning agent for (ab), 734
  - lesions other than meningiomas, visualization of tentorial branches of internal carotid artery in (ab), 1433
  - lesions, suspected, value of brain scans in management of (ab), 734
  - metastases, calcification in, 1249
  - tumors, contour brain scanning with iodine and mercury compounds for detection of, 177
  - tumors, preliminary evaluation of F^{18}-labeled tetrafluoroborate as scanning agent for (ab), 1442
- INTRAMURAL INTESTINAL HEMORRHAGE, 1061
- INTRAOSSEOUS VERTEBRAL VENOGRAPHY in diagnosis of lumbar disk disease, 1282
- INTRARENAL ARTERIOVENOUS FISTULA, congenital (ab), 486
- INTRAVASATION OF BARIUM, nonfatal, 1169
- INTRAVENOUS ANGIOCARDIOGRAPHY, abdominal aortography, and peripheral arteriography with single arm pressure injection, 893
- INTRAVERTEBRAL HERNIATIONS, difficulty of radiological diagnosis of certain (ab), 249
- INTUSSUSCEPTION in Western Nigeria (ab), 1214
- IODINE and mercury compounds for detection of intracranial tumors, contour brain scanning with, 177
- IRRADIATED PERSONS, accidentally, treatment of (ab), 253
- IRRADIATION by routine histological methods, assessment of response of cervical cancers to (ab), 974
  - in inoperable brochogenic carcinoma, clinical studies of combined chemotherapy and, 88
  - injury, acute accidental, value of bone marrow transplantation for (ab), 254
  - of lymphoma, two mev. wide field; twelve year experience, 124
- ISOTOPE TECHNIQUES, some medical, significance of photomultiplier fatigue in, 187

- JET STREAM phenomenon, ureteral, in adults, 341
- JOHN D. REEVES, JR., 1924–1964 (E), 223

- KIDNEY, congenital unilateral multicystic disease of; entity?, 281
  - left, torsion of spleen associated with congenital absence of (ab), 963
  - remaining, in irradiated survivors of Wilms's tumor, 148
- KIDNEYS, fetal lobulation of, 273
- KLINEFELTER'S SYNDROME, XXYY variant of (ab), 493
- KNEE, arthrography and arteriography in pigmented villonodular synovitis of, 1322

- LARYNGOGRAPHY, simple method of (ab), 712
- LARYNX and pharynx, contrast examination of; inspiratory phonation, 40
  - evaluation of clinical usefulness of roentgenologic findings of squamous cell carinomas of, 43
  - high voltage radiation: adjunct to surgery in large extrinsic malignancies of (ab), 249
- LATERAL FEMORAL CONDYLE, osteochondral fractures of (ab), 1217
- LEIOMYOMA of uterus, gas abscess within, 400
- LIEOMYOSARCOMAS, gastroduodenal: report of clinical, roentgenologic, and pathologic features of three surgical cases (ab), 246
- LEUKEMIA and lymphomas, chronic, colloidal zirconyl phosphate P^{32} in treatment of (ab), 253
  - medical radiations and; retrospective survey (ab), 736
- LEUKOPLAKIA constricting male urethra, intraluminal beta irradiation in (ab), 250
- LIPOMA, spinal, 1293
- LIVER and spleen, host-parasite relationship in human brucellosis with prolonged illness due to suppuration of (ab), 492
  - scanning and liver function tests: comparative study (ab), 253
  - splenoportography and portal vein thrombosis in patients with cirrhosis of (ab), 491
- LUMBAR discography (ab), 249
  - disk disease, intraosseous vertebral venography in diagnosis of, 1282
- LUNG and chest wall, spontaneous elimination of inhaled grass inflorescences through (ab), 1437
  - cancer: detection by use of electrical skin resistance method (ab), 713
  - simulated infiltration of, due to pleural thickening, 1373
  - environmental influences and carcinoma of (ab), 713
  - Hodgkin's disease of; roentgenologic-pathologic correlation, 96
  - polycystic: bronchiolar dilatation associated with muscular hyperplasia; emphasis on roentgenologic findings, 513
  - rust granuloma of: contribution to corroding, process of metallic splinters in body (ab) 1434
- LUNGS, radiological spectrum of aging and emphysematous (ab), 1436
- LYMPH NODES following interruption of their blood or lymphatic supply, influence of dual circulation on viability of (ab), 1220

LYMPHADENITIS, retroperitoneal tuberculous, 355
LYMPHADENOGRAPHY with radioactive contrast medium in retroperitoneal localization of malignant lymphogranuloma, 134
LYMPHANGIOGRAPHY: aid in urological surgery (ab), 971
  body distribution of ethiodol following (ab), 972
  chyluria: lymphatic-urinary fistula demonstrated by (ab), 972
  evaluation of its applications (ab), 725
LYMPHATIC DUCTS concomitant with neoplastic and systemic adenopathies, alterations of (ab), 724
LYMPHOEDEMA, chronic, in Western Nigeria (ab), 722
  praecox, venous obstruction in aetiology of (ab), 1219
LYMPHOGRANULOMA and pregnancy: prognostic and radiotherapeutic problems (ab), 485
  malignant, lymphadenography with radioactive contrast medium in retroperitoneal localization of, 134
LYMPHOGRAPHY in genital cancer (ab), 725
  in search for metastases, value of (ab), 492
  its place in urology (ab), 971
LYMPHOMA, two mev. wide field irradiation of; twelve year experience, 124
LYMPHOSARCOMA of digestive tract, Hodgkin's disease and (ab), 730

MAGNETIC FOREIGN BODIES from esophagus, stomach and duodenum with controllable permanent magnets, recent advances in removal of, 1021
MALE URETHRA, intraluminal beta irradiation in leukoplakia constricting (ab), 250
MAMMOGRAPHIC-PATHOLOGIC CORRELATION: calcification in breast disease, 29
MAMMOGRAPHY as routine screening examination for detecting breast cancer, 14
  nonmaligant variations in, 21
MARFAN SYNDROME and Abraham Lincoln (ab), 246
  skeletal manifestation in (ab), 246
MAXILLARY ANTRUM, intracavitary cobalt 60 beads in treatment of carcinoma of, 55
MEDICAL education in U.S.A., bicentennial of, 699
  radiations and leukemia; retrospective survey (ab), 736
(MEDULLARY VEINS), veins of white matter of cerebral hemispheres; diagnostic importance in carotid angiography, 739
MEGACOLON associated with administration of anticholinergic drug in patient with ulcerative colitis: report of case (ab), 715
MEGAVOLTAGE radiotherapy, osteosarcoma: study of value of preoperative (ab), 1222
MEGLUMINE iothalamate 60 per cent and sodium diatrizoate 50 per cent: comparison of urographic quality and side-effects (ab), 437
MELPHALAN in treatment of myelomatosis (ab), 1224
MENINGOCELE, intrathoracic, and neurofibromatosis (ab), 1212
MESENCHYMAL CHONDROSARCOMA (ab), 968
MESENTERIC ANGIOGRAPHY, indications for selective celiac and superior; experience with 128 cases, 1100
  artery, meandering, of colon, 1088
(MHP), 1-mercuri-2-hydroxypropane: new radiopharmaceutical for visualization of spleen by radioisotope scanning (ab), 732
MICROANGIOGRAPHIC STUDY of gastrointestinal lesions, 1173
MICROANGIOGRAPHY as technique; radiation effect versus artifact, 378
MITRAL DISEASE, posterior cardiac prominence in Ebstein's anomaly simulating, 1331
MORQUIO-ULLRICH DISEASE: new mucopolysaccharidosis (ab), 973
MOUTHGUARDS: protection against shock to head, neck and teeth (ab), 1432
MUSCULAR HYPERPLASIA, bronchiolar dilatation associated with: polycystic lung; emphasis on roentgenologic findings, 513
MYCOBACTERIAL DISEASES, 528
MYCOTIC INFECTIONS of bone (ab), 969
MYELOGRAPHY, air, cervical spondylotic myelopathy studied by, 1260
MYELOMA, of vertebral column, long-term survival of patients with (ab), 730
MYELOMATOSIS, melphalan in treatment of (ab), 1224
MYELOMENINGOCELE and spina bifida, urinary tract changes associated with, 255
MYOCARDIAL INFARCTION, calcification in coronary arteries and its relationship to arteriosclerosis and, 865
MYOSITIS OSSIFICANS as complication of tetanus (ab), 1218

NASOPHARYNGEAL chordoma (ab), 241
NATIONAL COMMITTEE ON RADIATION PROTECTION AND MEASUREMENTS (NCRP), 229
NATIONAL COUNCIL ON RADIATION PROTECTION AND MEASUREMENTS (NCRP), 951
NEAR-DROWNING, radiographic pulmonary changes in (ab), 1436
NECK OF FEMUR secondary to telecobaltherapy of pelvis, bilateral fracture of (ab), 250
NEOPLASMS, labeling of human globulin with I^{124} for positron scanning of (ab), 735
NEPHROTOXIC EFFECTS of oral cholecystographic agents, experimental investigation of (ab), 483
NEUROFIBROMATOSIS, intrathoracic meningocele and (ab), 1212
  spontaneous pseudarthrosis of tibia occurring in

patient with: report of case in man, forty-one years old (ab), 490
NUCLEAR RADIATION, recovery of male fertility after sterilization by (ab), 736

OLISTHETIC SCOLIOSIS (ab), 489
OPHTHALMIC ARTERIAL COMPLEX in angiographic diagnosis (ab), 1433
OPHTHALMO-MANDIBULO-MELIC DYSPLASIA; hereditary syndrome (ab), 965
OPTIC FORAMINA, radiologic study of (ab), 241
ORNITHOSIS; report of case, 1370
OSTEOBLASTOMA, benign: further observations and report of twenty additional cases (ab), 967
OSTEOCHONDRAL fractures of lateral femoral condyle (ab), 1217
OSTEOCHONDRITIS DISSECANS: treatment by bone grafting (ab), 1217
OSTEODYSTROPHY, renal glomerular (ab), 1440
OSTEOGENESIS IMPERFECTA at age of fifty-four, severely deformed patient with (ab), 248
  intrauterine, in four siblings (ab), 248
OSTEOLATHYRISM in rat: x-ray diffraction study of collagen from normal and lathyritic rats (ab), 493
OSTEOLYSIS after contusion. Three cases of osteolysis of acromial end of clavicle (ab), 1216
OSTEO-ONYCHO-DYSPLASIA, hereditary (ab), 1441
OSTEOSARCOMA of bone (ab), 247
  study of value of preoperative megavoltage radiotherapy (ab), 1222
OSTEOSCLEROSIS, generalized, secondary to metastatic medulloblastoma of cerebellum (ab), 969
  in chronic uremia (ab), 964

PAEDIATRIC RADIOLOGY, use of water-soluble contrast medium in (ab), 715
PAGET'S DISEASE, strontium 85 photoscanning in; with comments on several other skeletal problems, 195
PALATOPHARYNGEAL ROENTGENOGRAPHY, recent cinefluorographic advances in, 977
PANCOAST TUMOR, pitfalls in diagnosis of (ab), 242
PANCREAS demonstration of by roentgenography (ab), 482
PANCREATIC DISEASE, radioisotope photoscanning in diagnosis of, 1156
PANVENTRICULOGRAPHY: new method with emulsified pantopaque (ab), 1433
PARARENAL PSEUDOCYSTS; report of 3 cases, 286
PARASITIC INFESTATIONS, pulmonary manifestations of (ab), 1435
PARATHYROID scanning with selenium 75 labeled methionine (ab), 1223
  tumors, adaptability of selective angiography in localization of (ab), 1212
PARIETOGRAPHY, an additional study of stomach (ab), 1213
PATELLOFEMORAL EROSION and synovial hypertrophy, association of: diagnostic entity (ab), 490
PELVIC PNEUMOGRAPHY, 390
PELVIS, bilateral fracture of neck of femur secondary to telecobaltherapy of (ab), 250
PERCUTANEOUS transfemoral arteriography; analysis of complications in 1,000 consecutive cases, 918
  transhepatic cholangiography; preliminary report describing new approach to examination, 1131
PERICARDIAL effusion by radioisotope heart scanning, detection of (ab), 252
  fluid correlated with I^{131}-cholografin and IHSA heart scan, measurement of (ab), 252
PERIPHERAL ARTERIOGRAPHY, intravenous angiocardiography, and abdominal aortography with single arm pressure injection, 893
PEYRONIE'S DISEASE, effect of x-ray therapy on patients with (ab), 975
PHARYNX, contrast examination of larynx and; inspiratory phonation, 40
PHOTODENSIMETRIC ANALYSIS of tomographic image (ab), 494
PHOTOMULTIPLIER FATIGUE in some medical isotope techniques, significance, of, 187
PINEAL LOCALIZATION, new method of, 1242
PITUITARY GLAND, chromophobe adenomas of, 160
PLEURAL THICKENING, simulated infiltration of lung due to, 1373
POLAROID UROGRAPHY: useful adjunct (ab), 718
PORTAL vein gas: grave prognostic sign, 1162
  vein thrombosis in patients with cirrhosis of liver, splenoportography and (ab), 491
  venous thrombosis, calcification in portal venous system: unusual radiologic sign of (ab), 491
PORTOCAVAL ANASTOMOSIS for cirrhosis, intrahepatic portal thomboses following (ab), 491
POSITRON SCINTILLATION CAMERA for detecting simulated brain tumors with gallium 68-EDTA, sensitivity of, 174
PNEUMOCYSTIS CARINII PNEUMONIA and congenital hypogammaglobulinaemia (ab), 1435
PNEUMOGRAPHY, pelvic, 390
PNEUMO-MEDIASTINOGRAPHY by trans-sternal method (ab), 1437
PNEUMOPERITONEUM in infancy and childhood (ab), 484
  progressive postoperative; air entering through drain sites, 1166
PNEUMOSTRATIGRAPHY, frontal, of cerebellar hemispheres (ab), 1432
PREGNANCY and lymphogranuloma: prognostic and radiotherapeutic problems (ab), 485
  late, studies of inferior vena cava in (ab), 484
PRELIMINARY PROGRAM: sixty-fifth annual meeting of American Roentgen Ray Society, 225
PRESIDENT'S ADDRESS; first a radiologist, 1427
PRIMITIVE TRIGEMINAL ARTERY as seen angiographically and at postmortem examination, 761

PROSTATE, radiographic estimation of size of (ab), 1441
PROSTIGMINE, accelerated transit through small bowel after (ab), 481
PSEUDOARTHROSIS, spontaneous, of tibia occurring in patient with neurofibromatosis: report of case in a man, forty-one years old (ab), 490
PSEUDOPHEOCHROMOCYTOMA (ab), 965
PULMONARY ALVEOLAR PROTEINOSIS; roentgenologic analysis, 571
  artery, main, segment following pulmonary embolism, changes in, 557
  cavitation in Wegener's granulomatosis, 521
  changes in decompression sickness: some observations on compressed air workers at Clyde tunnel (ab), 243
  edema: correlation of roentgenologic appearance and abnormal physiology, 495
  embolism by radiating microspheres, diagnosis of (ab), 960
  embolism, changes in main pulmonary artery segment following, 557
  hemosiderosis, nodular aspirational, 561
  infection and hidden tumor (ab), 242
  insufficiency and fluorodensimetry, air trapping, 501
  manifestations, acute dermatomyositis presenting with (ab), 960
  manifestations of parasitic infestations (ab), 1435
  metastasis, massive, report of case with: so-called adamantinoma of long bones (ab), 247
  metastasis, solitary, "benign" giant-cell tumor of bone with: case report (ab), 968
  neoplasm, clinical observations during and following radiotherapy in patients with (ab), 730
  neoplasms, complications and after effects of high energy radiation therapy of (ab), 974
  sequestration, intralobar, angiographic diagnosis of, 540
  thromboembolism—case for pulmonary angiogram (E), 693
PYCNODYSOSTOSIS (ab), 966
PYELOGRAPHY, drip infusion: indications and applications in urologic roentgen diagnosis (ab), 1440
  infusion, 1391
  intravenous, with 66.8 per cent sodium iothalamate; Conray-400, iodine content 40 per cent, note on, 267
PYELONEPHRITIS, chronic, ureteral manifestations in; roentgen diagnostic study, 329

RADIATION cataract in clinical therapeutic radiology (ab), 976
  effect versus artifact; microangiography as technique, 378
  injuries, acute local, treatment of (ab), 254
  injury to right hand, accidental, occupational (ab), 729
  therapy, bone sarcomas following (ab), 731
  therapy, eustachian tube obstruction and (ab), 975
  therapy for malignant lesions in region of pelvis, bone changes following (ab), 732
  therapy, usefulness of roentgen shielding pastes in low voltage (ab), 729
RADICAL TREATMENT, should more be attempted in breast cancer? Caldwell Lecture, 1963, 3
RADIOACTIVE contrast medium in retroperitoneal localization of malignant lymphogranuloma, lymphadenography with, 134
  isotopes, device for interstitial adminstration of, 64
RADIODIAGNOSTIC techniques, quantitative ratio of requisite radiation to available information in different (ab), 493
RADIOGRAPHIC aspect of Cooley's disease in adult, considerations on (ab), 1218
  estimation of size of prostate (ab), 1441
  pulmonary changes in near-drowning (ab), 1436
  teaching file, building a, 448
RADIOISOTOPE heart scanning, detection of pericardial effusion by (ab), 252
  photoscanning in diagnosis of pancreatic disease, 1156
  renal scanning in assessment of renal disease, role of (ab), 718
  renography, acute renal failure; diagnosis of etiology by (ab), 717
  scanning, 1-mercuri-2-hydroxypropane (MHP): new radiopharmaceutical for visualization of spleen by (ab), 732
RADIOLOGIC anatomy of ileocecal value (Bauhin's valve) (ab), 481
  examination of colon with spasmolytic drug given by rectum (ab), 481
  examination with pneumoperitoneum, laparoscopy and hepatic biopsy: differential diagnosis of "large irregular liver" by "semiologic triad" (ab), 1215
RADIOLOGICAL EXAMINATION of two pairs of craniopagus twins (ab), 726
RADIOPAQUE CATHETER, selective transtracheal bronchography using, 591
RADIOTHERAPEUTIC PROBLEMS, lymphogranuloma and pregnancy: prognostic and (ab), 485
RADIOTHERAPY in patients with pulmonary neoplasm, clinical observations during and following (ab), 730
RADIUM THERAPY in cancer of eyelid (ab), 1222
RECTOSIGMOID, electrolyte depletion syndrome associated with villous adenomas of, 1123
RECTUM, radiologic examination of colon with spasmolytic drug given by (ab), 481
RED CELL AGGREGATION from concentrated saline and angiographic media (ab), 970
REEVES, JOHN D., JR., 1924–1964 (E), 223

- RENAL arteriography, percutaneous noncatheter left brachial, 315
  - arteriography, upright, in study of renal hypertension (ab), 486
  - artery, atrophic, 309
  - artery lesions in renovascular hypertension, modification and extension of aortography for evaluation of, 321
  - disease, role of radioisotope renal scanning in assessment of (ab), 718
  - failure, acute, angiography in (ab), 486
  - failure, acute: diagnosis of etiology by radioisotope renography (ab), 717
  - hydatid disease (ab), 486
  - hypertension, performance of divided renal function tests in: practical method (ab), 717
  - normal size: absolute criterion, 270
  - size, roentgenologic demonstration and evaluation of reduction in (ab), 716
  - tubular damage, hypercalcuric rickets associated with (ab), 964
  - variants, some normal, 1380
- RENOVASCULAR hypertension, curable, value of urea washout test in assessment of, 1397
  - modification and extension of aortography for evaluation of renal artery lesions in, 321
- RESIN SPONGE MODIFICATION of I^{131} T_3 test (ab), 251
- RETICULOSES, disseminated herpes zoster in, 116
- ROENTGEN carcinoma 20 years after irradiation of thoracic wall sarcoma (ab), 729
  - injuries, combination, after contact therapy of skin carcinoma (ab), 729
  - radiation and ultrasound, combined, results of treating malignant skin tumors with (ab), 250
  - shielding pastes in low voltage radiation therapy, usefulness of (ab), 729
- ROENTGENOGRAPHIC demonstration with water-soluble iodine contrast medium, experimental infarction of small bowel in dogs, 1080
  - features of common ventricle with inversion of infundibulum; corrected transposition with rudimentary left ventricle, 652
  - features of congenital corrected transposition of great vessels; comparative study of 33 cases with roentgenographic classification based on associated malformations and hemodynamic states, 623
- ROENTGENOLOGIC demonstration and evaluation of reduction in renal size (ab), 716
  - demonstration of an air-fluid level, preoperative diagnosis of brain abscess by; report of two cases, 844
  - studies, physiology of ureteral obstruction as determined by, 291
- ROENTGENOGRAPHY, demonstration of pancreas by (ab), 482
- SAHARA FLUORINE and its aggressiveness, origin of; dental lesions in chronic fluorosis (ab), 1432
- SARCOMA, Ewing's (ab), 730
  - primary cardiac, successful sterilization of, by supervoltage radiation therapy, 942
- SCANNING AGENT for intracranial lesions, gallium-68 as (ab), 734
- SCIENTIFIC EXHIBITS, American Roentgen Ray Society, 1413
- SCINTIGRAPHY, quantitative estimation of spleen size by (ab), 736
- SCOLIOSIS, olisthetic (ab), 489
- SCROTUM, calcified steatomata of; report of case, 388
- SECTION ON INSTRUCTION, American Roentgen Ray Society, 457
- SELENIUM 75 labeled methionine, parathyroid scanning with (ab), 1223
- SILICATOSIS, sign of: multiple progressive calcific pleural plaque formation (ab), 1437
- SINGLE ARM PRESSURE INJECTION, intravenous angiocardiography, abdominal aortography, and peripheral arteriography with, 893
- SIXTY-FIFTH ANNUAL MEETING of American Roentgen Ray Society (E), 218, (E), 1410
  - instruction courses of (E), 455
  - preliminary program, 225
- SKELETAL TUBERCULOSIS—1963 (ab), 1216
- SKIN CARCINOMA, combination roentgen injuries after contact therapy of (ab), 729
- SMALL bowel after prostigmine, accelerated transit through (ab), 481
  - bowel diseases, unusual; methods and observations, 1072
  - bowel in dogs, experimental infarction of; roentgenographic demonstration with water-soluble iodine contrast medium, 1080
  - intestinal diverticulosis, 1048
- SODIUM IOTHALAMATE 66.8 per cent (conray-400), clinical evaluation of, 676
  - note on intravenous pyelography with; Conray-400, iodine content 40 per cent, 267
- SPASMOLYTIC DRUG given by rectum, radiologic examination of colon with (ab), 481
- SPINA BIFIDA and mylomeningocele, urinary tract changes associated with, 255
- SPINAL cord, multiple lesions of, 1298
  - lipoma, 1293
- SPINE, tumors of and their relationship to intervertebral disk; roentgenographic-anatomic correlation, 1270
- SPLEEN associated with congential absence of left kidney, torsion of (ab), 963
  - by radioisotope scanning, 1-mercuri-3-hydroxypropane (MHP): new radiopharmaceutical for visualization of (ab), 732
  - hamartoma of; angiographic observations, 1150
  - size by scintigraphy, quantitative estimation of (ab), 736
- SPLENOMEGALY, osseous Gaucher's disease in early

childhood: report of case with extensive bone changes and pathological fractures without (ab), 488
SPLENOPORTOGRAPHY and portal vein thrombosis in patients with cirrhosis of liver (ab), 491
SPONDYLOLYSIS and spondylolisthesis following low back fusions (ab), 969
SPRAIN FRACTURE, recent isolated, roentgen diagnosis of; fractured ulnar styloid, 1307
SQUAMOUS CELL CARCINOMA of larynx, evaluation of clinical usefulness of roentgenologic findings in, 43
STEATOMATA, calcified, of scrotum; report of case, 388
STEREOGRAPHIC cineangiocardiography and strereoscopic fluoroscopy (ab), 973
STEREOSCOPIC fluoroscopy and stereographic cineangiocardiography (ab), 973
STOMACH and duodenum with controllable permanent magnets, recent advances in removal of magnetic foreign bodies from esophagus, 1021
  glomus tumor of; case report, 1026
  parietography, an additional study of (ab), 1213
STREPTOKINASE, thrombolytic treatment of acute occlusion with (ab), 1219
STRONTIUM 85, diagnosis of bone metastasis by photoscanning with (ab), 735
  for detection of bone lesions, use of (ab), 1223
  photoscanning in Paget's disease; with comments on several other skeletal problems, 195
SUBARACHNOID HAEMORRHAGES, recurring spontaneous rupture of urinary bladder following (ab) 1440,
SUBCLAVIAN ARTERY, aberrant right; dispute of commonly accepted concepts, 855
  associated with cervical rib, poststenotic dilatation (aneurysm) of; report of two cases, 1328
SUBDURAL EMPYEMA; report of case demonstrating unusual angiographic triad, 838
SUPERIOR VENA CAVAL OBSTRUCTION, esophageal varices in, 1000
SUPERVOLTAGE RADIATION THERAPY, successful sterilization of primary cardiac sarcoma by, 942
SUPRASELLAR TUMORS, evaluation of angiography in diagnosis of, 811
SYNOVIAL HYPERTROPHY, association of patellofemoral erosion and: diagnostic entity (ab), 490
SYPHILIS, report on certain recently observed cases of early congenital bone (ab), 487

TEACHING FILE, radiographic, building a, 448
TECHNICAL EXHIBITS, American Roentgen Ray Society, 1423
TELECESIUM THERAPY, grid irradiation in (ab), 250
TELECOBALTHERAPY of pelvis, bilateral fracture of neck of femur secondary to (ab), 250
TELECOBALT UNITS, on specification of field size for, 207
TELETHERAPY UNIT, double headed cobalt 60, 202
TELEVISION SYSTEMS, image intensifier, performance evaluation of, 418
TERATOMA in infant, gastric tridermal (ab), 714
TESTICULAR TUMORS, germinal, 152
TETANUS, myositis ossificans as complication of (ab), 1218
THORACIC AORTA, arteriosclerotic aneurysms of; intravenous and selective visualization, 1353
  duct in man, cineroentgenographic examinations of (ab), 723
  duct in some cardiopathies, first observations on morphologic alterations of (ab), 724
  duct with 60 opacifications in living human, anatomo-radiologic study of (ab), 723
  wall sarcoma, roentgen carcinoma 20 years after irradiation of (ab), 729
THORIUM DIOXIDE (thorotrast) deposits in human unstained bone narrow smears, recognition and clinical importance of (ab), 733
THOROTRAST GRANULOMAS, 584
THROMBOEMBOLISM, pulmonary,—case for pulmonary angiogram (E), 693
THROMBOTIC OCCLUSION of abdominal aorta; seven year angiographic study of case, 887
THYROID GLAND, autofluorography of, by means of image amplification (ab), 734
TOMOGRAPHIC IMAGE, photodensimetric analysis of (ab), 494
TONGUE, complete oral reconstruction in conjunction with cobalt 60 teletherapy for carcinoma of; case report, 59
  in identical twins, carcinoma of, 213
TRACHEOBRONCHIAL COMPRESSION due to vascular sling (ab), 243
TRANSCRICOTHYROID BRONCHOGRAPHY, some observations on, 578
TRANSHEPATIC CHOLANGIOGRAPHY (ab), 962
  percutaneous (ab), 1439
TRANS-STEENAL METHOD, pneumo-mediastinography by (ab), 1437
TRANSTRACHEAL BRONCHOGRAPHY, selective, using radiopaque catheter, 591
TRANSVERSE COLON, herniation of, through foramen of Morgagni simulating left ventricular hypertrophy, report of case, 1013
TRICUSPID VALVE, Ebstein's anomaly of; angiocardiographic considerations, 1338
TRIOLEIN-I^{131} tolerance in patients with coronary heart disease (ab), 732
T-tube cholangiogram deformities, cinecholangiography as aid in interpretation of (ab), 962
TUBERCULOSIS, hematogenous pulmonary, rapid clearing in (ab), 712

skeletal,—1963 (ab), 1216
TUBERCULOUS lymphadenitis, retroperitoneal, 355

ULNAR STYLOID, fractured; roentgen diagnosis of recent isolated sprain fracture, 1307
ULTRASOUND AND ROENTGEN RADIATION, combined, results of treating malignant skin tumors with (ab), 250
UREA WASHOUT TEST in assessment of curable renovascular hypertension, value of, 1397
UREMIA, chronic, osteosclerosis in (ab), 964
URETER, endometriosis of; rare manifestation of common disease, 351
URETERAL jet stream phenomenon in adults, 341
  manifestations in chronic pyelonephritis; roentgen diagnostic study, 329
  obstruction as determined by roentgenologic studies, physiology of, 291
  varices, 346
URETHROGRAPHY, retrograde, by flexible sound with balloon (ab), 1216
URINARY bladder, choice of contrast material and possibilities of double contrast visualization of (ab), 1216
  bladder following subarachnoid haemorrhages, recurring spontaneous rupture of (ab), 1440
  tract changes associated with spina bifida and myelomeningocele, 255
UROGRAPHIC QUALITY and side-effects, meglumine iothalamate 60 per cent and sodium diatrizoate 50 per cent: comparison of (ab), 487
UROGRAPHY, double dose contrast medium in (ab), 717
  excretory, in azotemic patient: revised technique (ab), 716
  excretory, improved method of (ab), 963
  polaroid: useful adjunct (ab), 718
UROLOGIC complications following irradiation of carcinoma of uterine cervix (ab), 251
  roentgen diagnosis, indications and applications in: drip infusion pyelography (ab), 1440
UROLOGICAL surgery, lymphangiography: aid in (ab), 971
UROLOGY, lymphography: its place in (ab), 971
UTERINE CERVIX, urologic complications following irradiation of carcinoma of (ab), 251
UTERUS, gas abscess within leiomyoma of, 400

VASCULAR and intravascular responses to contrast media, influence of low molecular weight dextran on, 682
  insufficiency of lower extremities, serial arteriography in (ab), 721
  sling, tracheobronchial compression due to (ab), 243
VEIN OF GALEN, aneurysm of; case report and review of literature, 756
VENOUS OBSTRUCTION in aetiology of lymphoedema praecox (ab), 1219
VENTRICLE, rudimentary left, corrected transposition with; roentgenographic features of common ventricle with inversion of infundibulum, 652
VENTRICULAR HYPERTROPHY, left herniation of transverse colon through foramen of Morgagni simulating; report of case, 1013
VERTEBRAL and basilar arteries causing cranial nerve involvement, case of aneurysmal malformation of, 836
  column, long-term survival of patients with myeloma of (ab), 730
VERTICOSUBMENTAL ROENTGENOGRAM in cephalometrics (ab), 1212
VISCERAL HETEROTAXY with malformed heart: similar findings in child and puppy (ab), 961
  injuries in battered children (ab), 972
VITAMIN-D resistant rickets: long-term clinical study of eleven patients (ab), 1217

WATER-SOLUBLE contrast medium in paediatric radiology, use of (ab), 715
  iodine contrast medium, roentgenographic demonstration with; experimental infarction of small bowel in dogs, 1080
WEGENER'S GRANULOMATOSIS, pulmonary cavitation in, 521
WILMS'S TUMOR, remaining kidney in irradiated survivors of, 148

X-RAY apparatus, archaeology with (ab), 737
  dosages to general population of United States, weighted gonadal diagnostic roentgen-ray doses in teaching hospital with comments on, 404
  film, commercial, effect of uniform gamma ray fogging on diagnostic usefulness of (ab), 727
  films and screens, frequency response characteristics of, 426
  films in diagnosis of chronic amoebic hepatitis (ab), 482
  therapy on patients with Peyronie's disease, effect of (ab), 975
X-RAYS in detection of calcium in biological tissues at microlevel, use of (ab), 973
XXYY variant of Klinefelter's syndrome (ab), 493

AUTHOR INDEX TO VOLUME 92

*=Original Article

Abrams, Herbert L., *1150
Accomazzi, Franco, 1218
Adams, Herbert D., 1212
Adesola, A. O., 1214
Adler, Denis C., 959
Akkary, S., 712
Alberti, G. P., 724
Alfidi, R. J., 488
Alpert, Meyer, 247, 731, 1441
Amar, Arjan D., 717
Amberg, J. R., *1128
Amplatz, Kurt, *1397
Anderson, A. E., Jr., 1436
Anger, Hal O., *174
Ansfield, F. J., *88
Arbeit, Sidney, *850
Ardran, G. M., 1434
Ariel, Irving M., *64, 960
Aronow, S., 1442
Aronow, Saul, 734, 735
Ash, C. L., *202
Ashley, Franklin L., *977
Athanasiades, Th., 1437
Atkinson, H. R., 736

Baert, A. A., 249
Bagshaw, Malcolm A., *942
Bain, A. D., 961
Baird, R. J., 1219
Baker, Hillier L., Jr., 1433
Bandirali, G. R., 487
Barden, Robert P., *495
Barker, Kirby G., *341
Baroncelli, G., 730
Barr, M. L., 493
Barry, William F., Jr., *1301
Barry, William J., *584
Barth, E., 482
Bates, Lloyd M., *426
Batt, Richard C., 456
Beabout, John W., *855
Beadenkopf, William G., *865
Becker, Melvin H., 241
Beckett, J. M., 1434
Beentjes, Lucas B., *404
Behar, Albert, 726
Beierwaltes, William H., 1223
Beller, Harry E., 969
Bellucci, Richard J., *844
Berk, Leonard, 490
Berlin, Leonard, *351
Bernstein, D. C., 1439
Bery, K., 967
Best, Edward B., 223
Bickel, William H., 719
Bilodeau, Larry P., *1322
Birch, John R., 962
Birkhead, Newton C., 962
Bishop, Robert C., 716
Bismuth, V., 481, 720
Black, Samuel P. W., 969
Blendis, L. M., 1437
Bliss, B. P., 1440
Bloedorn, Fernando G., *77
Bloom, A., 1214
Blumenfeld, J. Isidoro, 1218
Bodian, Martin, 1435
Bohatirchuk, F., 973
Boldrey, Edwin B., *160
Bonte, J., 1216
Bookstein, Joseph J., 492
Bosniak, Morton A., *850
Bottini, E., 1220
Boucher, B. J., *116
Bourisiquot, P., 1438
Boyarsky, Saul, *291
Bräutigam, Hildegard, 250
Breed, J. Ernest, 1222
Brenner, George H., *390
Bret, P., 714
Brewer, R., *302
Brody, H. Stephen, 971
Brown, Michael W., *1255
Brown, T. C., *96
Brownell, Gordon L., 734
Brummett, Stanley W., *977
Brunton, F. J., 1438
Buchanan, J., *676
Bücheler, E., 714
Buermann, August, *1282
Bujdoso, Laszlo, 971
Bulls, Antonio A., *213
Burhenne, H. J., 714
Burhenne, H. Joachim, 715
Burke, Gerald, *1156
Burnett, L. L., 976
Burris, John O., *1338
Burrow, Gerard N., 1221
Bush, Leonard F., 968
Butt, W. P., 249
Byrne, John J., *1131

Cabassa, N., 1443, 1444
Cabrera, Aurelio, 247, 730, 968
Calnan, J. S., 1219
Cameron, John R., 253
Caplan, Lawrence H., *1048
Capotorti, L., 1220
Capretti, G., 730
Carbonara, Peter, 1441
Carey, Lewis S., *623, *652
Carlson, Harley C., *989
Carpender, J. W. J., 727
Carr, D. H., 493
Carruthers, Bruce M., 732
Carter, C. Conrad, *769
Casarett, George W., *378
Ceccarelli, Frank E., 489
Cev, Muzaffer, 1213
Charkes, N. David, 252, 735
Chase, Norman E., 1433
Chaumont, P., 720
Chawla, S., 248, 967
Chen, Shek C., *55
Cheng, Tan Ban, 241
Chepey, Julius J., *55
Cherry, C. P., 974
Chevrot, L., 1441
Chiappa, S., *134
Chick, Ernest W., 969
Chitour, M., 1441
Choremis, C., 1437
Chrenka, Paul, 247
Christoforidis, Anthimos J., *513, *595
Ciembroniewicz, Julius, *838
Cimmino, Christian V., *281
Claessins, H., 1216
Clark, William G. B., 1440
Cohen, Donald M., 730
Cole, C., *302
Collard, M., 712
Collica, Carl J., *192
Concannon, J. P., *302
Cooley, Robert N., *404, 693
Coomaraswamy, R. P., 491
Cooperman, Lawrence R., *273, *1123
Copestake, G. G., 727
Corbelle, G., 491
Corcoran, Cornelia, 735
Correa, Roy J., Jr., 963
Cortes, Octavio, 1433
Cossu, F., 724
Covington, E. Eugene, *213
Cowley, R. Adams, *77
Crandall, Paul H., *1260
Craven, J. D., 1440
Croppi, Carlos J., 481, 1215
Crosca, A., 966
Crow, Neil E., 718
Cuccia, Carlo A., *77
Cunningham, J. R., *202
Currarino, Guido, *1312
Currie, Donald J., 962
Currie, J. C. M., 486

Dagradi, A. E., 1214
Dahlin, David C., 719, 730

Dalrymple, Glenn V., 734
Daoud, Assaad S., *865
Dardari, M., 1215
Daves, Marvin L., *390
Davidson, John K., 243
Dayan, A. D., *116
Deaver, J. Montgomery, 962
Debray, Ch., 481
Delahaye, R. P., 1438
Del Guerico, L. R. M., 491
Dent, C. E., 964
DePriest, Oscar S., III, *1013
Desgrez, H., 1216
Di Chiro, Giovanni, *441
Diethelm, L., 1216
DiGiulio, Walter, 1223
Di Marco, G., 1215
Diner, Wilma C., *390
DiSalvo, Nicholas A., 1212
Djilali, G., 1441
Dolan, Patrick A., 725
Doppman, John L., 490, *1380
Doran, M. L., 1219
Dotter, Charles T., 487
Dowling, Edmund A., 968
Doxiades, T., 482
Drummond, B. J., *1021
Du Boulay, G. H., 726
Dubowitz, Lilly M. S., 960
Dubowitz, Victor, 960
Dudley, H. Robert, 247
Duggan, H. E., 975
Dunbar, F. Scott, 243
Duquesnel, C., 714
Duquesnel, J., 714
Dursin, C., 1216

Eckert, Phillippa, 1436
Edmunds, L. H., Jr., *1100
Elebute, E. A., 1214
Eliasoph, J., *1061
Elkin, M., 491
Elkin, Milton, *291
Elkington, S. G., 1439
Ellis, Kent, *669, *1338
Ennis, Leroy M., 242
Entzian, W., 1442
Epperson, Robert D., *1192
Evans, John A., *267, 961

Faegenburg, David, *355
Falchi, Q., 250
Fam, Aziz, 963
Feddema, J., 493
Feinberg, S. B., *1275
Feindel, William, *177
Feiring, Emanuel H., *811
Felson, Benjamin, *1088
Fendley, Claude E., *591
Fenn, John E., 1213
Ferencz, Charlotte, 961
Ferguson, George B., 249
Ferrand, J., 1441
Ferrante, E., 1220
Ferris, Ernest J., *838, *1131
Fiasconaro, Joseph, 1212
Finby, Nathaniel, *309, 246
Finch, Stuart C., 733
Fink, Howard E., Jr., 483
Finney, Louis A., *1282
Fischer, J., 736
Fleming, Richard J., *1338
Fletcher, Gilbert H., *43
Flocks, R. H., *255
Fons, Stanley D., *152
Forfar, J. O., 961
Fossati, F., 728
Foster, W. D., *302
Fred, Herbert L., *521
Freiberger, Robert H., 487
Friedman, M., 964
Fries, James F., 713
Frischbier, H. J., 492
Fu, Whei-Rung, *341
Fujii, Masamichi, *378
Fuller, Roger H., 1436

Galli, G., *134
Galton, D. A. G., 1224
Gambill, Earl E., 491
Gandini, D., 959
Gargano, Fredie P., *1282
Gaudino, Fulvio, 1218
Gayarre, Carlos Gil, 241
Gayarre, Miguel Gil, 241
George, David L., *1188,*1192
Gilchrist, Milton, *1373
Gilroy, John, 948
Giraud, M., 714
Glenn, William W. L., 1213
Glucksmann, A., 974
Goidanich, I. F., 973
Goldman, H. I., 712
Goldstein, M. S., *1156
Gollin, F. F., *88
Gollin, Frank F., 253
Gondos, Bela, *329
Goodman, J. R., 1214
GopalaRao, U. V., *426
Gordon, Sewell S., *1373
Goree, John A., *829
Gosselin, Arthur J., *872
Gottschalk, Alexander, *174
Grabiger, Rudolf, 729
Grabstald, Harry, *365
Graf, R. A., *255
Graham, Andrew J., 1213
Gras, F., 730
Green, Robert A., *561
Greenberg, Lowell H., 1224
Greenberg, S. Donald, *521
Greene, Margaret W., 735
Greenough, William G., *994
Greville, Nicholas R., 1217
Griffiths, D. Ll., 248
Griffiths, Sylvia P., *669, *1338
Gunz, F. W., 736
Gutkin, Morton L., *844
Guyard, G., 1216

Haag, Edmund L., *341
Haastert, S., 1439
Haber, Seymour, *1029
Hagstrom, Jack W. C., *877
Halaby, Fouad A., 725, *1293
Hale, John, *1188
Hall, Eric J., *207
Halpern, Mordecai, *918, *1353
Hamilton, Howard B., 1221
Hanafee, William, 486
Hanafee, William N., *1260
Hanson, Daniel J., *1331
Harris, John H., *1391
Harris, John H., Jr., *1391
Harrison, C. V., 974
Harrison, Edgar G., Jr., *29
Harrison, William E., Jr., 488
Haskin, Marvin E., *557
Haupt, George J., 962
Haverback, Bernard J., 959
Heetderks, Dewey R., Jr., 716, 963
Hegeman, Jane G., *187
Heiskell, C. L., 1214
Heitz, F., 1216
Helff, John R., 971
Hemley, Samuel D., *309, *578
Herbert, Walter W., *907
Heslin, D. James, *1166
Hiemeyer, V., 1219
Hildebrand, H., 245
Hill, J. M., 253
Hillman, David C., *1013
Hinck, Vincent C., *769
Hodges, Fred J., III, *1255
Hodges, Philip J., *215
Hohenfellner, Rudolf, 251
Holm, Thure, *418
Holmes, R. Brian, 722
Holt, Earl P., Jr., 959
Holtz, S., *40
Hoover, Norman W., 719
Hope-Stone, H. F., *116
Hotchkiss, Robert S., 736
Howard, John M., 1220
Hrubec, Zdenek, 1221
Huang, Yun Peng, *739, *792
Hudak, A., 481
Hughes, Robert R., 725
Hugues, A., 7[illegible]3

Hurley, Edward, *942

Irwin, Gerald A. L., *1169
Iwata, Ryuzaburo, 482

Jackson, David M., *776
Jackson, Frank I., *688
Jacobson, Harold G., *1048
Jacox, Harold W., 241
Jaffee, Henry L., 735
Jameson, A. Gregory, *669
Jansen, Cornelius R., 735
Jesse, Mary Jane, *669
Jesseph, John E., 735
Jesserer, H., 1212
Jewell, James H., 968
Johanson, Paul, *1322
John, H. E., *202
Johnson, Carl E., 485
Johnson, E. R., *1128
Johnson, Philip M., *1139
Joison, Julio, *1131
Jolly, R., 1438
Jones, Walter C., III, 1216
Joyce, John J., III, 967
Judkins, Melvin P., 487

Kaindl, F., 1212
Kalliana, P., 966
Kambolis, Catherine P., *1270
Kamener, R., 712
Kandt, D., 1434
Kanick, Virginia, *309, *578
Kaplan, Neville, *291
Kastner, Jacob, 734
Katerelos, H., 1437
Katz, Sol, 712
Kaufman, Joseph H., 486
Kaufman, Joseph J., *346
Kay, H. E. M., 254
Kaye, Josse, 712, 960
Keats, Theodore E., 969
Keiser, D. v., 492
Keller, Richard H., *528
Kemp, F. H., 1434
Kerr, M. G., 484
Khilnani, M. T., *1061
Kim, Seuk Ky, *1301
Kincaid, Owings W., 491, *855
Kirsh, David, 969
Kiser, William S., 717
Kisseler, B., 482
Kittredge, Richard D., *309
Klein, Edward W., *195
Klein, Rubin, *43
Klinefelter, Edmund W., *1307
Koehler, P. Ruben, 972
Kogure, Takashi, *1017
Köhler, R., 721
Kohn, P., 1212
Konstam, Peter, 722
Kotscher, E., 1212
Kountz, S., 1219
Krabbenhoft, K. L., 1410
Kreel, L., 1437
Kreuzer, H., 965
Kroening, Paul M., *1370
Kundel, Harold L., 492
Kupperman, Herbert S., 718
Kurohara, Samuel S., *378
Kurtzman, Raymond S., *1000
Kuttig, Helmut, 250

Laaban, J., 1438
Lachapèle, A. P., 723
Lagarde, Cl., 723
Lampe, William T., II, 970
Lapides, Jack, 963
Larsen, Parry B., 1213
Larson, Sanford J., *786
Lascano, Raul, 481
Laurenson, Rae Duncan, 720
Lawrence, John H., 1222
Lawson, Edward A., 1432
Lear, Philip, 960
Leaver, Robert C., *1293
Lee, E. Stanley, 1222
Leeds, Norman, 1433
Lees, F., 248
Lefebvre, J., 720
Leger, Lucien, 491, 1218
Leicht, E., 244
Leistner, G. H., 482
Lemaigre, G., 491
Le May, Marjorie J., *776
Lembeck, F., 244
Lenczner, M., 1435
Lenzi, Luciano, 973
Lessel, Wilhelm, 729
Levin, Joseph, 718
Levitan, Leonard H., *29
Lichtenstein, Louis, 967
Light, John P., *615
Linberg, Eugene J., *77
Linfoot, John A., 1222
Ling, J. T., *315, *676
Lippincott, Stuart W., 735
Loeb, Ellen, 253
Lofstrom, James E., *55
Loken, Merle K., *1397
Lombardi, G., *1298
Loop, John W., *1072
López, Waldo, *888
Louis, Herbert J., 488
Love, Blanche M., *865
Love, Leon, *786
Low, Ira H., *418
Lowman, Robert M., *273, *1123
Luborsky, F. E., *1021
Lund, Ronald R., *195
Lynch, Edward C., *521

McAdams, George B., 251
McAfee, John G., 732
McAlister, William H., *375, *1173
McCandless, Robert G., *1162
McClintock, James T., 734
McCort, James, 972
McDonald, Eugene J., 221
McIntosh, Hamish W., 732
McIntyre, D. J., 976
McKhann, C. F., *1100
McLain, Clarence R., Jr., *351
McRae, Donald L., *177
McWhirter, Robert, *3
MacDonald, J. C. F., 727
MacEwan, D. W., 243
MacEwen, K. F., *96
Mackenzie, D. H., 1222
MacKinnon, Kenneth J., 486
MacLeod, John, 736
Macleod, W., 961
Malt, Ronald A., *1166
Mangum, Jack F., 726
Mankin, Henry J., 490
Margulis, Alexander R., *1080, *1173
Marinaro, Alex, *426
Marrocu, F., 724
Marshak, R. H., *1061
Marshall, Thomas R., *315, *676
Marshall, Victor F., 972
Marshall, W. C., 1435
Martinez, Jose, *291, 732
Massaro, Donald, 712
Mathé, Georges, 253
Maurer, Hans-Joachim, 713
Maxwell, Morton H., *346, 486
May, Ivan A., 242
Mayer, Victor, 487
Mazzanti, G., 974
Mendelsohn, E. A., 718
Mercado, Raul, Jr., *77
Meyer, Maurice W., 970
Meyers, Harvey I., 959
Meyers, Wayne A., 972
Miller, H., 491
Miller, R. A., 961
Miller, Roscoe E., *448
Miller, Wallace E., 1216
Millick, P., 966
Mittelpunkt, Allen, *786
Mitty, Harold A., *355
Mnaymneh, Ghandur, 247
Mnaymneh, Walid A., 247
Möbius, G., 244
Moldenhauer, W., 1213
Moon, Alfred C., 242
Moore, Booker T., *187

Moran, John M., 1221
Morgan, H. G., *116
Morgan, Russell H., *426
Mori, Paul A., 1436
Morse, Dryden, 251
Mosca, Lidio G., 481, 1215
Moseley, Robert D., Jr., *418
Moskowitz, Myron, *1088
Mosser, Donn G., 455
Mouktar, Marcel, 1218
Mouton, Ramon A., 484
Movitt, Eli R., 726
Moynihan, F. J., 248
Murphy, David R., 243
Murphy, J. A., 247
Murray, J. F., 960
Murtland, Richard L., *1139
Myers, Richard N., 962

Nahoum, Henry I., 1212
Naito, Seiji, 482
Naji, A. F., 247
Nathan, M. H., 1436
Nebesar, R. A., *1100
Nehrkorn, Otto, 729
Nelson, Sidney W., *513, *595
Neville, W. E., 247
New, Paul F. J., 965
Newton, T. H., 721
Newton, Thomas H., *756
Ngu, V. Anomah, 722
Nice, Charles M., Jr., 484
Nielsen, P. Birger, *547
Norman, Alex, *1270
North, Luceil B., *1042
Noyek, Arnold M., *844

Obel, I. W. P., 712
Ochsner, Seymour Fiske, 1200
Oderr, Charles, *501
Odom, Guy L., *829
Oettinger, Leon, Jr., 735
Ogden, Ralph T., *152
Ogura, J., *40
Oliva, L., 494
Oliver, John A., 486
Oon, C. L., *1242
Ortiz, Federico, 972
Oster, Walter F., *615
Otis, Richard, *152
Otto, Donald L., *1000

Pachter, M. R., 731
Pandolfo, G., 966
Paolaggi, J.-A., 481
Parker, R. G., 976
Pasquinelli, V., 485
Passerini, A., *1298
Pastorelle, D. Joseph, Jr., *192
Patel, J.-Cl., 491
Patt, N. L., *96
Patterson, Richard J., 719
Paul, Sheila Singh, 966
Pearson, J. B., 963
Penta, Arthur Q., *1021
Pentecost, B. L., 1219
Perey, Daniel Y. E., 243
Perl, John I., 250
Peterson, Harold O., 218, *1227
Peterson, Richard B., *1293
Petrocini, S., 959
Phelan, John T., 247, 730, 968
Phillips, Elbert W., *388
Phillips, Theodore L., *160, 731
Pichard, R., 481
Pierce, Robert H., 964, *1232
Piercy, J. E., 1437
Pierotti, P., 250
Pignataro, E., 494
Pillay, V. K., 488, 965
Pinet, A., 1432
Pinet, F., 1432
Pinniger, J. L., 486
Pitts, Neal C., 1218
Plastina, U., 966
Plunkett, E. R., 493
Pollard, J. J., *1100
Pollard, James J., 965
Pollycove, Myron, 726
Pomeranz, Maurice M., *21
Ponseti, Ignancio V., 1217
Poole, Graham, 242
Porot, J., 1216
Porter, William R., 726
Portera, Alberto, 1433
Potts, D. Gordon, *1249
Powers, W. E., *40
Pratesi, A. Cavina, 487
Pratt, Philip C., *513, *595
Pochaczevsky, Rubem, *365
Profirov, Dimiter, 729
Pye, Theo H., *341

Rachmilewitz, Eliezer, 726
Rae, A. I., 486
Rai, Kanti, 735
Rall, Kenneth L., *286
Ramirez-R., Jose, *571
Ramsay, Andrew J., *215
Ramsay, G. Craig, *1338
Ranniger, Klaus, *540
Rao, P. L., 966
Rapp, Robert, 963
Rapp, W. J., 1216
Read, Raymond C., 970
Redd, Bryan L., Jr., 732
Reeder, Maurice M., *1013
Reeves, Robert J., 975
Reichel, Gerhard, 729
Reid, Bevan L., 733
Rein, Burton I., *321, *1322
Reinck, H., 1213
Reinermann, Theodor, 729
Reinfrank, Ralph F., 251
Reingold, I. M., 1214
Reuter, S. R., 721
Rhangos, William C., 969
Richter, Curt P., 713
Ricketts, Howard J., 1213
Ricketts, James, 1432
Ricketts, Robert M., *977
Righi Riva, G. C., 1215
Rimondi, C., 974
Rippey, John G., *769
Robinson, Richard G., 1212
Roenigk, William J., 483
Rogers, Lee F., 484
Roland, Arnold S., 960
Rominger, C. Jules, *584
Rosenbaum, Harold D., *1331
Rosenbaum, Howard T., 1436
Rosenberg, J. C., 246
Rosenberg, Norman J., 1217
Rosenthall, Leonard, 252, 718, 1223
Ross, Gilbert, Jr., *286
Roviralta, Emilio, 714
Rowe, George, 973
Roze, R., 730
Rubenfeld, Sidney, *192
Rubin, Ira C., *850
Rubin, Philip, *378
Rucker, T. N., 488
Runyon, Ernest H., *528
Russell, Walter J., *1017
Russell, Warren, *756
Ruttenberg, Herbert D., *623, *652

Sagerman, Robert H., *942
Saito, Toshio, 482
Salzman, Ferdinand A., *124
Samuel, Eric, 484
Sanchez, Francisco R., 243
Sanders, D. E., 1435
Santino, A., 1215
Sargent, Thornton, 1222
Saunders, Michael, 1440
Sawyer, Wayne R., 967
Sayago, Roberto, 481
Scharer, Lawrence L., 715
Schencker, Bernard, 1440
Scherr, David D., 248
Schiavi, G. F., 485
Schmidt, Robert M., *877
Scheider, Harold J., *10[illegible]6
Schneider, Louis, 1437

Schoefer, G., 1434
Schoop, W., 1219
Schwartz, David T., 247
Schwartz, Harold, 246
Schweinsberg, Michael, 1441
Scott, D. B., 484
Scott, Michael, *836
Scuro, L. A., 724
Segall, Harold N., 244
Seidenberg, Bernard, *850
Seitzman, David M., 725
Severini, A., *134
Shaffer, Burton, 972
Shaldon, Stanley, 486
Shapiro, Jerome H., *811, *1131
Shapiro, Robert, *1380
Shea, John, 962
Shealy, C. Norman, 734
Sheline, Glenn E., *160, 731
Sherrick, D. W., 491
Sheville, Eli, 486
Shillingford, J. P., 1219
Shipps, Fred C., *935, *939
Shuler, Stanon E., 966
Sidaway, Muriel E., 715
Sigmann, Joan M., 492
Simon, Allan L., *270
Simon, Harold, 242
Sims, J. LeRoy, 253
Sitterson, Beecher W., 736
Skelley, Joseph F., 972
Sklaroff, David M., 252, 735
Sloan, Robert F., *977
Smedal, Magnus I., *124
Smith, J. H., *255
Snider, James R., 718
Soloway, A. H., 1442
Soltan, H. C., 493
Sommer, Leonard S., *872
Sondheimer, Fred K., *1110
Spaulding, W. B., 1435
Speed, Dorothy E., 1224
Speer, R. J., 253
Spink, Wesley W., 492
Spira, Errol N., 244
Spjut, Harlan J., *1173
Stacy, Gilbert S., *1072
Staple, Tom, *375
Starr, Paul, 735
Stasney, R. J., 247
State, D., 491
Staub, Edward V., *1397
Stauffer, Henry, 1222
Stauffer, Herbert M., *836
Stearns, Genevieve, 1217
Stein, Günter, 250
Stein, Harry L., *893
Steinberg, Israel, *267, *877, *893, *1110, *1328, *1353
Steinberg, Stanley B., *557
Steiner, R. E., 1219
Stejskal, Robert E., *1397
Stempien, S. J., 1214
Stenger, John M., 1432
Stergiou, L., 482
Stewart, Bruce H., 716
Stewart, James R., *855
Stolberg, H. O., *96
Stradling, Peter, 242
Strax, Philip, *21
Stubbins, S. Gaines, *59
Sturm, A., Jr., 965
Subirana, M., 1432
Sulis, E., 724
Sullivan, C. Roger, 721
Summers, R. E., *302
Svare, Gerhart T., *1245
Svien, Hendrik J., 730
Svoboda, M., 245
Swan, A., 1224
Sweet, R. D., 254
Sweet, W. H., 1442
Sweet, William H., 735
Symmonds, Richard E., 721

Tanaka, Kouichi, 1224
Tapia, Jorge, 1217
Templeton, Arch W., *591, *1255
Teplick, J. George, *557
Ter-Pogossian, Michel, 734
Theodorou, S., 1437
Thomas, Martin A., 221
Thomas, M. Lea, 486
Thomas, R. Glyn, 712
Thompson, Ian M., *286
Thompson, William L., 1436
Thomson, F. B., 245
Thorfinnson, P. Carmelle, 722
Thurber, Deloran L., *14
Thurn, P., 716
Tilak, S. P., 1220
Tilos, Felicia, *400
Tindall, George T., *829
Tøjner, Henning, 489
Trinkle, J. Kent, 717
Tristan, Theodore A., *1192
Trump, John G., *124
Tsitsanis, D., 482
Turano, Luigi, *986
Turk, Arthur, *192
Turner, J. W. Aldren, 243

Vaeth, Jerome M., *148
Valenzuela, German Abad, 1215
Valvassori, Galdino E., *540, 964, *1232
Vandervelde, Gerald M., *989
Van de Velde, E., 1216
Van Epps, E. F., *255
Vasselle, B., 1216
Vaudagna, James, 972
Verhaak, R. L. O. M., 714
Vermes, Edmund, *844
Vermooten, Vincent, 1441
Vermund, H., *88
Vest, Bruce, *1080
Vest, Thomas Bruce, 734
Viallet, P., 1441
Viamonte, Manuel, Jr., *872
Viljanen, V., 721
Vita, G., 1216
Vrousos, C., 1432

Wagner, Henry N., Jr., 732
Waldman, Irvin, *1312
Waldman, Irving, *351
Walker, John Hunt, *67
Walter, L., 974
Warwick, O. H., *96
Walzak, Myron P., 972
Weaver, D. S., 1214
Weaver, Kenneth, *1139
Wee, Lee Khee, 965
Weens, H. Stephen, *1042
Weghaupt, Karl, 251
Weijer, D. L., 975
Weil, C., *302
Weiner, Irwin M., 732
Weintraub, Herbert D., *286
Weintraub, Ronald A., *400
Weissleder, H., 723, 1219
Wellens, P., 249
Weston, H. J., 1435
Westover, Joseph L., *977
Wexler, Lewis, *1150
Wheeler, Edward C., 1433
White, Fletcher H., *351
Whitesel, John A., 971
Wiedeman, Mary P., *682
Wiens, Ruth G., 493
Wilner, Harvey I., 246
Wilson, Carl L., 718
Wilson, George P., 483
Wilson, Morton C., 718
Wimpfheimer, Felix, 1437
Winckelmann, G., 1219
Winter, Chester C., 717
Witten, David M., *14, *29
Wizenberg, Morris J., *77
Woeber, Karlheinz, 250
Wolf, B. S., *1061
Wolf, Bernard S., *739, *792
Wolf, R., 736
Wollschlaeger, Gertraud, *761, *1252
Wollschlaeger, Paul B., *761, *1252

Wootton, P., 976
Worde, Boyd T., 975
Wright, Jack M., 1432
Wright, Kenneth A., *124

Yamamoto, Y. Lucas, *177

Yiotsas, Z., 482
Ypey, J., 737

Zanca, Peter, *341
Zanelli, Joan, *177
Zarnstorff, William C., 973
Zatzkin, Herbert R., *1169
Zboralske, F. F., *1128
Zeitler, E., 1216
Zimmerman, Harold, *1088
Zizmor, Judah, *844
Zuber, O., 244
Zwi, S., 712

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ADRENAL PHEOCHROMOCYTOMA

Removal of Right-Sided Adrenal Pheochromocytoma

In 1959, during examination for recurrent headaches, the patient, a 38-year-old man, was found to have hypertension.

Routine medical and urologic studies showed no abnormalities. However, patient was admitted for thorough evaluation when blood pressure rose to 190/140 mm of mercury and the headaches continued.

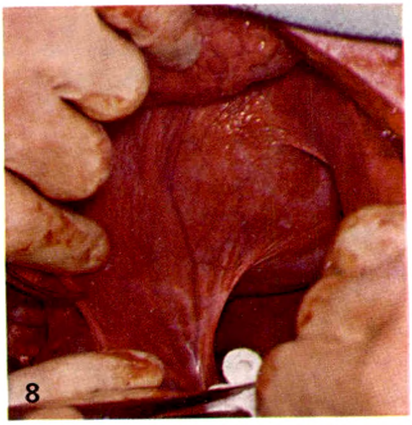
Physical examination, including electrocardiogram and radiographic studies of chest and kidneys, showed no abnormality (Figure 1).

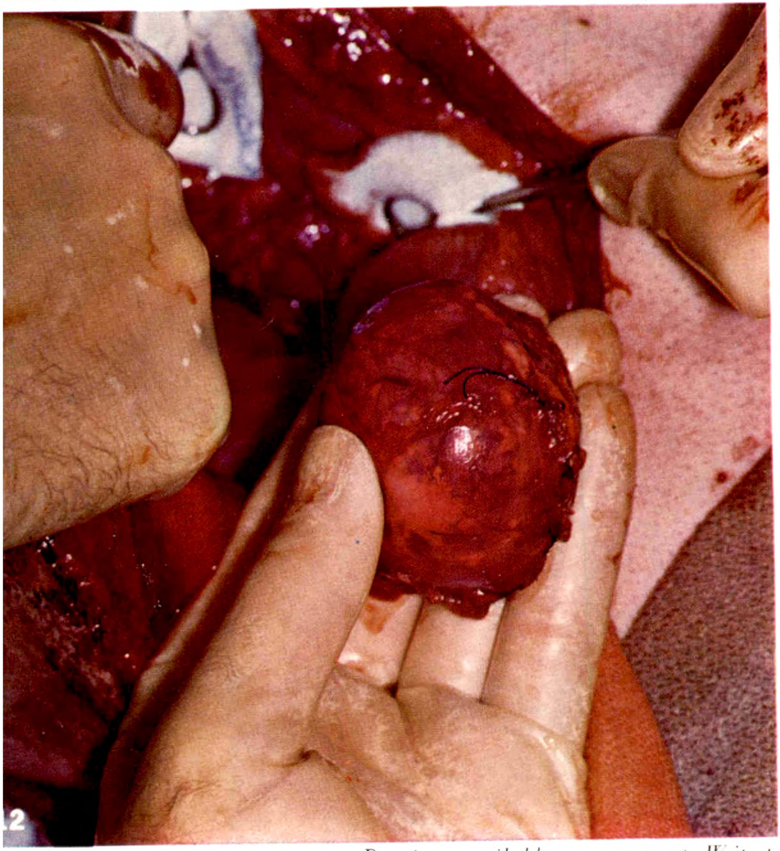
Phentolamine test and chemical studies, including measurement of vanillyl mandelic acid (VMA) and of urinary excretion of catechol amines, indicated a *pheochromocytoma*.

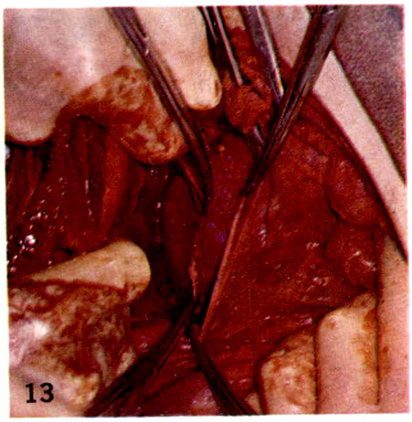
Retroperitoneal gas insufflations combined with radiographic and tomographic studies confirmed and localized the pheochromocytoma in the right adrenal gland (Figures 2 and 3). Following surgery, patient's blood pressure returned to normal, where it has remained.

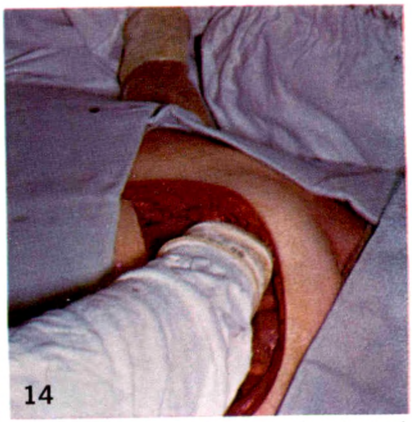
Diagnostic radiographs and steps in the ensuing surgery follow.

7


8


2


13


14

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Adrenal Pheochromocytoma

(CONTINUED)

Figure 4. Intra-arterial catheter is needed to monitor blood pressure in case of sudden hypotension or peripheral vascular constriction, and permits rapid infusion of corrective medications.

Figure 5. Patient is hyperextended on the operating table to permit better access to adrenal fossae behind the ribs.

Figure 6. Paracostal incision is outlined. This can be extended in either direction.

Figure 7. This incision permits retraction of liver upward and medially and intestines downward and medially.

Figure 8. Posterior peritoneum is incised over upper end of right kidney.

Figure 9. Right kidney is retracted downward and the 5 cm adrenal tumor can be seen just above renal vein, and very close to it. See drawing.

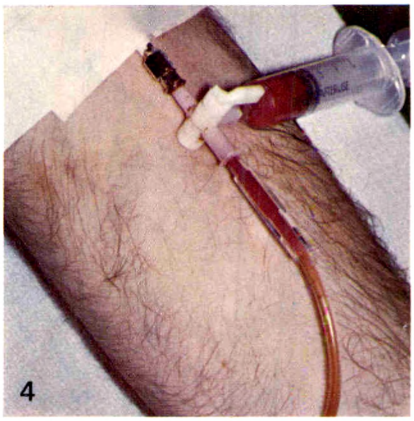
Figure 10. Tumor has been freed around its lateral borders and from renal vein below by meticulous dissection.

Figure 11. Tumor is elevated and vessels running medially from it into the aorta and vena cava are divided and ligated individually.

Figure 12. Tumor is lifted from its bed above kidney and shows a rim of adrenal cortex extending around it as a yellow, caliper-like line.

Figure 13. Cut edges of the peritoneum are closed over kidney without drainage.

Figure 14. Opposite adrenal as well as area along the aorta and vena cava down beyond the bifurcation is explored for presence of additional tumors.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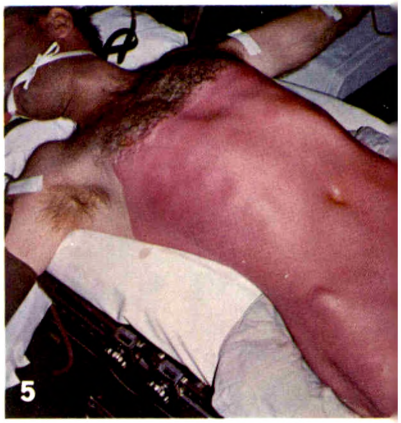
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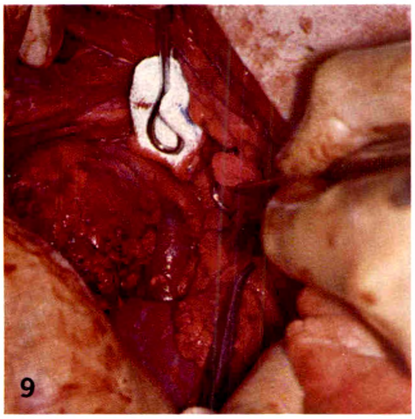
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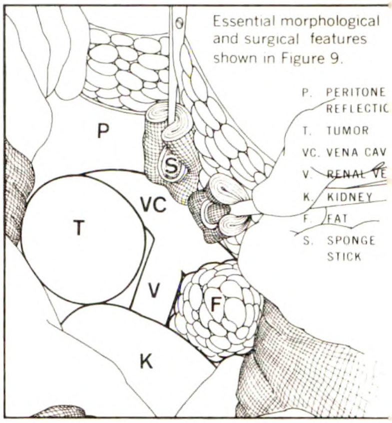
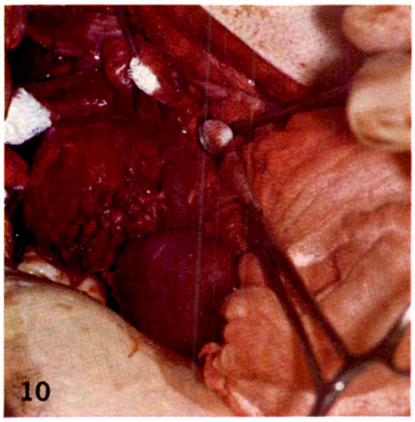

Essential morphological and surgical features shown in Figure 9.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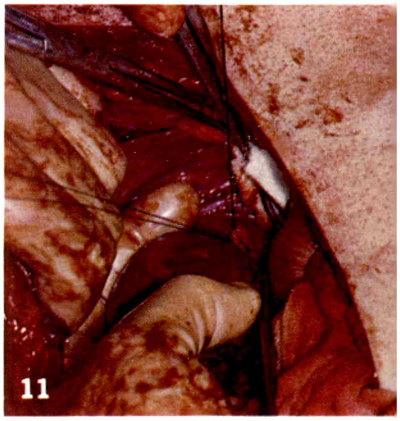
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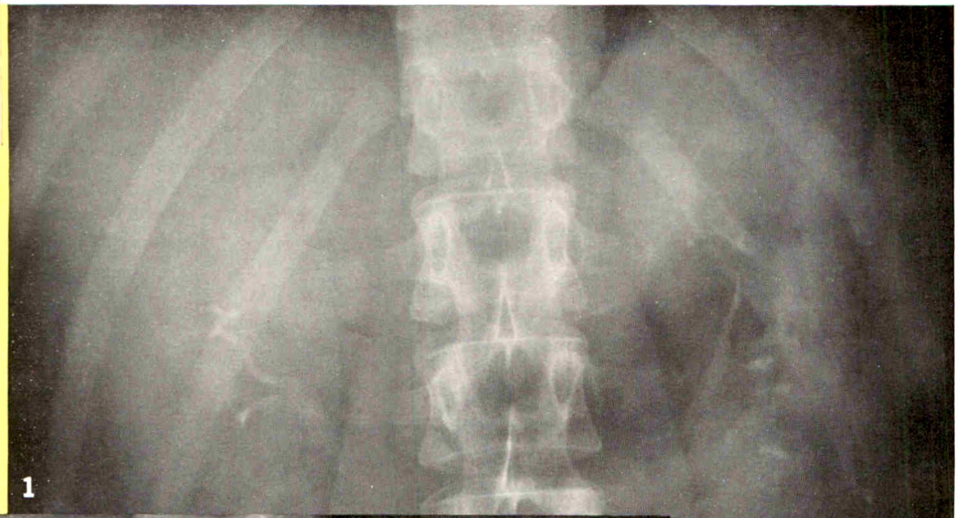
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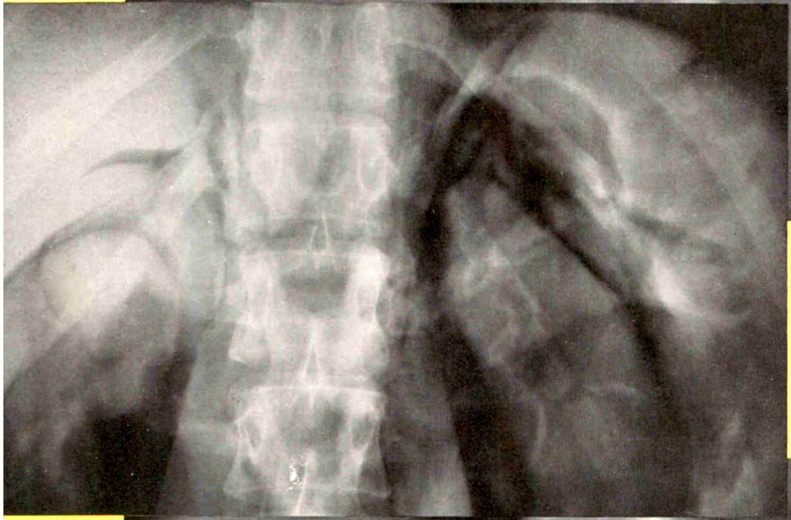

Figure 2. Retroperitoneal gas insufflation clearly defines the margins of both kidneys and left adrenal gland. These are normal in appearance. Radiograph, however, clearly shows a rounded mass in the area of the right adrenal displacing the normal triangular adrenal shadow superiorly.

aphic study
eal gas insuf-
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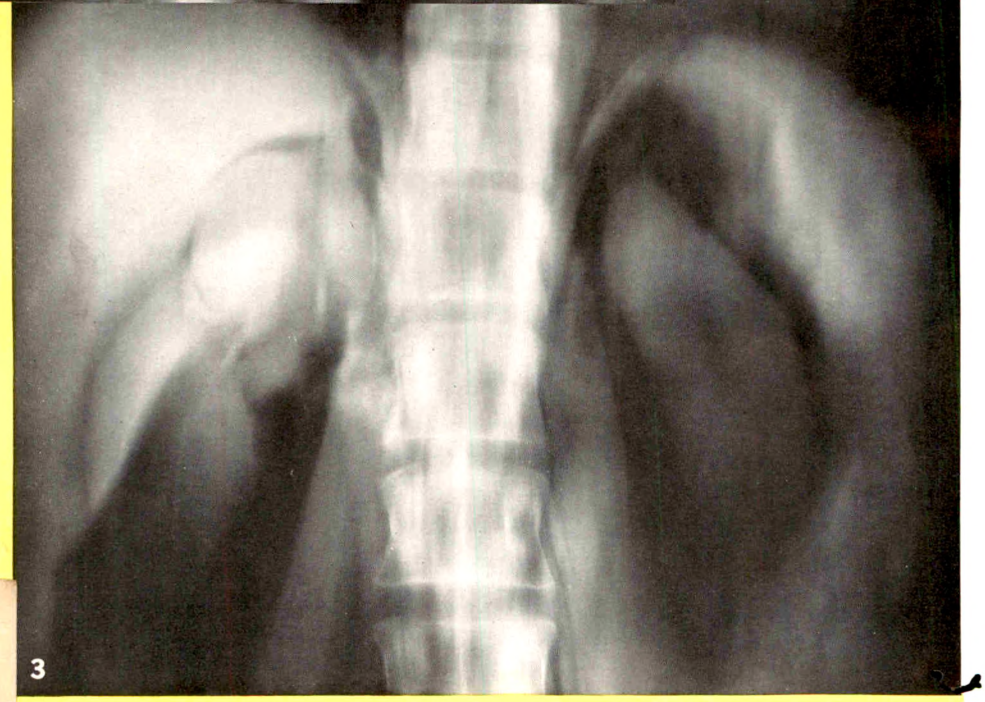
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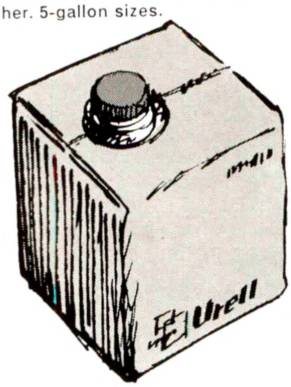

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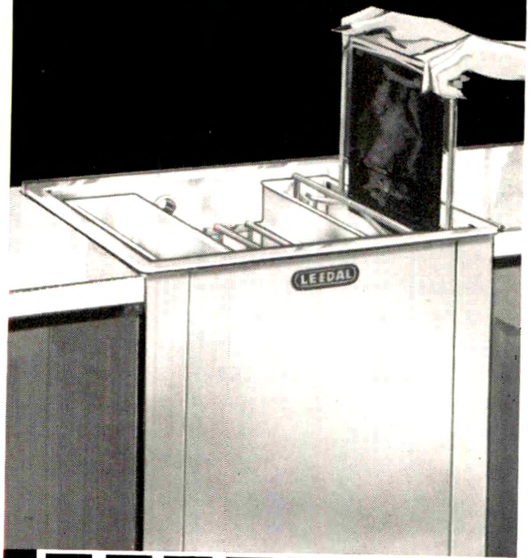

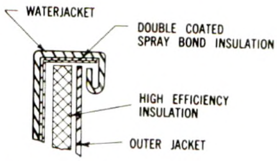
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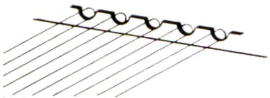

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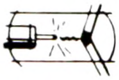

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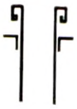

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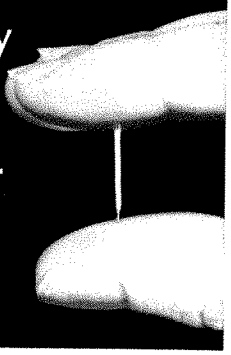

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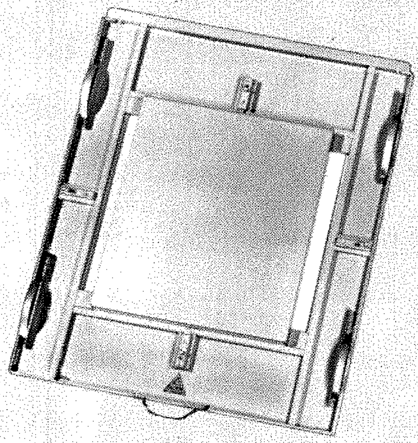

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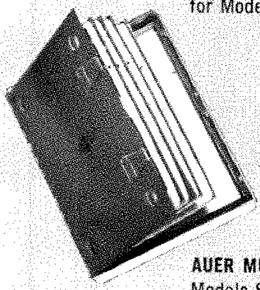

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Ansco (General Aniline & Film Corp.) xxxix
Atomic Energy of Canada Ltd. xxxv
Automatic Seriograph Corp. (Division of Litton Industries, Inc.) xxvi

Barnes-Hind Barium Products xviii
Bell-Craig, Inc. xxviii

Cenco X-Ray Corporation xvi, xvii
Continental X-Ray Corporation xl
Controls for Radiation, Inc. li

Dunlee Corporation xix
du Pont, E. I. de Nemours Co. x, xi

Eastman Kodak Company xxvii, xlv, xlvi
E-Z-Em Company xx

Fougera, E., & Company, Inc. vi

General Electric Company xxix, xxx, xxxi, xxxii, xli

Halsey X-Ray Products viii
Hanley Medical Equipment Company l
Hunt, Philip A., Chemical Corporation liv

Ilford, Inc. xxi

Karger, S. AG xlvii
Keleket X-Ray Corporation ix

Lafayette Pharmacal, Inc. xxxvi
Leedal, Inc. xlix
Liebel-Flarsheim Company (Division of Ritter Co) xii
Low X-Ray Corporation xiii

McElroy, Donald, Inc. xlviii

North American Philips Company xxiv, xxv
N. V. Philips Gloeilampenfabrieken xxxviii
Nuclear-Chicago Corporation xv

Pako Corporation liii
Physicians Technical Equipment Company li
Picker X-Ray Corporation xxiii, xlii, Fourth Cover
Profexray, Inc. i, Third Cover

Schick X-Ray Company xiv
Siemens Medical of America, Inc. xxxvii
Squibb, E. R., & Sons v, xxxiv
Standard X-Ray Company xxii

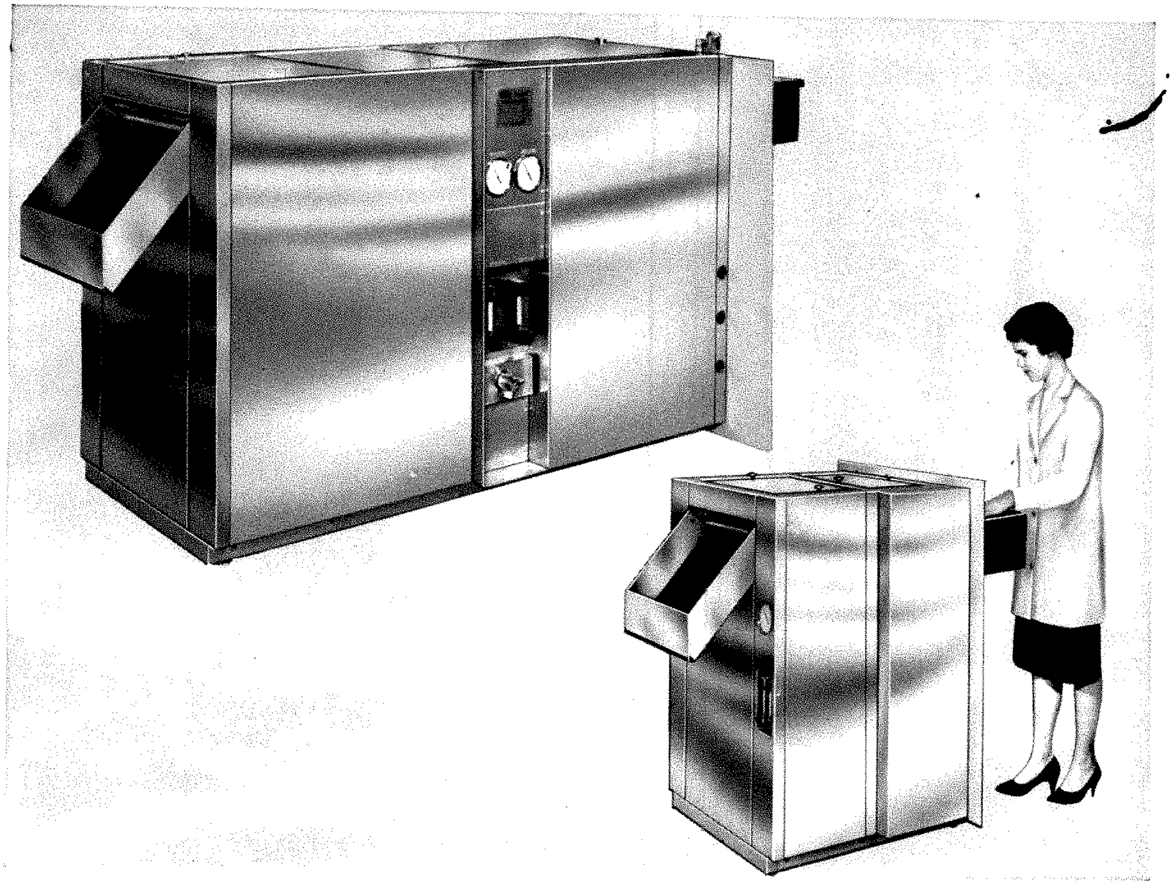
Thomas, Charles C, Publisher xliv, l

U. S. Radium Corporation xliii
Urell, Inc. xlviii

Westinghouse Electric Corporation Second Cover
Winthrop Laboratories xxiii
Wolf X-Ray Products, Inc. vii

●

We try to present an accurate index. Occasionally this may not be possible because of a last-minute change or an omission.



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